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# HEMIPLEGIA IN CYANOTIC CONGENITAL HEART DISEASE

BY

BERYL CORNER AND BRUCE PERRY

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Received May 22, 1942

Although the cases to be described lack any anatomical observations, the association of uncomplicated cyanotic congenital heart disease with the sudden onset of hemiplegia appears worthy of record. All three cases have only recently come under our observation.

## NOTES OF CASES

*Case* 1.—J. L. was first seen at the age of eight months. He was a first child, and because of the mother's "kidney trouble" the pregnancy was terminated three weeks early. Shortly after birth it was noticed that the hands and feet went blue at times and that when crying he appeared to get unduly out of breath. He had gained weight satisfactorily. On examination there was no marked cyanosis and the only clinical abnormality was a loud central systolic murmur. On X-ray, there was a heart shadow suggesting the tetralogy of Fallot. A month later he was admitted to Southmead Hospital because, following a particularly intense attack of cyanosis and dyspnoea five days before, it was noticed that the right arm and leg did not move normally and that on crying the face was drawn over to the left side. On admission there was a right hemiplegia. The cardiac physical signs were unchanged, there was no fever, and no other abnormality was found. He continued to have attacks of cyanosis of varying severity. A lumbar puncture a week after admission was blood-stained—apparently traumatic—but otherwise normal. A blood count showed 6,000,000 red cells with a haemoglobin of 90 per cent. Since then there has been no change.

*Case* 2.—D. B. was born normally and was found to have congenital heart disease at the age of three months. He progressed favourably except for attacks of cyanosis until the age of eleven months, when loss of power in the right arm and leg was noticed a week after an attack of bronchitis. He was admitted to hospital the next day and found to have a right hemiplegia. The cerebrospinal fluid two days after the onset showed 6 lymphocytes per c.mm. and 50 mg. of protein per 100 c.c. Ten days later this was normal. There

was a mild pyrexia for three weeks. Since then the child has remained the same. There is constant cyanosis with exacerbation in attacks. There is dextrocardia with a basal systolic murmur. X-ray examination shows a boot shaped heart with dextrocardia. Electrocardiogram shows right axis deviation when allowance is made for the dextrocardia. A recent blood count shows 9,000,000 red cells with a haemoglobin of about 160 per cent. This is probably a case of the tetralogy of Fallot with dextrocardia.

*Case* 3.—M. W. was first seen at the age of nine years. He is deeply cyanosed with a soft basal murmur. X-rays show a rather boot shaped heart with a small pulmonary arc. His electrocardiogram shows marked right axis deviation. He has 7,000,000 red cells per c.mm. and a haemoglobin of 140 per cent. The X-ray picture suggests that this may be a case of Fallot's tetralogy but the murmur is so soft as to raise doubts about this diagnosis. When first seen he had a right hemiplegia which was at first regarded as congenital. However, on further enquiry his mother states that this was first noticed at the age of eight months, after a week's illness in which he "seemed dazed."

## Discussion

This syndrome was entirely new to us, and the only references found in the literature are the cases reported by Ford (1937) and the case recently reported by Wood (1942).

Ford's first case was a mentally defective child dying at the age of two and a half of pneumonia. He exhibited athetoid movements at the age of twelve months and the colour is said to have been "dusky." Post-mortem, there was a patent ductus arteriosus and a patent interventricular septum. The left middle cerebral artery showed an old thrombosis, partly recanalized, with an infarct involving the motor cortex and part of the temporal lobe and extending into the internal capsule and lenticular nucleus.

His other case was a child, blue from birth, who at ten months showed a basal systolic thrill and murmur and a red cell count of 7·5 millions. At two years of age he suddenly became stuporous and developed a left hemiplegia. A few days later he became more comatose and showed a bilateral extensor plantar response. He died shortly after. Post-mortem, there was atresia of the pulmonary artery, patent interventricular septum patent foramen ovale, and cardiac hypertrophy. There was a thrombosis of the right middle cerebral artery with infarction, and a later thrombosis of the superior longitudinal sinus and a small infarct involving part of the left motor cortex.

Wood's patient had been cyanosed from birth. There was no definite history of the onset of hemiplegia, but this apparently developed in infancy, since she had never walked and did not sit up until two years of age. Post-mortem, there was no gross vascular lesion but degenerative changes apparently due to the intense congestion. Bodechtel (1932) has described changes in the brain in congenital and acquired heart disease, apparently due to anoxaemia, but these changes would hardly appear sufficient to produce hemiplegia.

Ford attributes these thromboses to the polycythaemia and compares this with the cerebral thrombosis that may complicate polycythaemia vera in the adult. However, in our first case the polycythaemia was slight, and in Ford's first case the cardiac malformation should have produced little cyanosis except with the terminal pneumonia.

Since Wood's case showed no actual occlusion it appears that the brain lesion associated with cyanotic congenital heart disease may be produced in at least two ways. Obviously the possibility of embolism must be considered, but it would appear likely that if this were the cause, emboli elsewhere would have been observed. The possibility of a superimposed bacterial endocarditis seems to be excluded by the subsequent history. The actual pathology of the cases here reported must remain uncertain.

## Summary

Three cases of cyanotic congenital heart disease developing sudden hemiplegia in infancy have been described. The possible mechanism of this has been discussed.

We are indebted to Dr. R. M. Norman for drawing our attention to the observations of Bodechtel.

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# CONGENITAL PULMONARY ATRESIA WITH CEREBRAL THROMBOSIS AND HEMIPLEGIA

BY

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*From the Cardiac Department of the London Hospital and the Bernhard Baron Institute of Pathology*

Received July 7, 1942

In view of the recent communication of Corner and Bruce Perry (1942) the following case is of special interest.

A girl, aged 19 (Fig. 1), was first seen on February 21, 1942, when she was admitted to the London Hospital for dental extractions. Since the age of 2 she had been under observation for " heart trouble " with blueness and swollen fingers. Her activities had always been limited by shortness of breath, although for the last three years she had been capable of light work in the home. There had been no faints or cerebral attacks, and there was no history of congenital heart disease in parents or siblings.

She was a tall and well-developed girl with purple cyanosis; this was general, but most striking on the cheeks and lips, as well as in the fingers and toes, which were grossly clubbed. The pulse was regular and of good volume. The apex beat was palpable in the fifth space in the mid-clavicular line, and there were no thrills. Normal heart sounds were heard in all areas, and there was a diffuse systolic murmur of only moderate intensity, maximal in the pulmonary area and below it. The blood pressure was 120/80. There were scattered rhonchi over both lungs and slight lumbar œdema, but no other signs of heart failure.

Radiological examination showed much enlargement of the cardiac outline to the left, probably of the right ventricle, as it was of a *sabot* shape. There was a deep concavity in the pulmonary artery region and only slight hilar shadows (Fig. 2). The blood count showed: red cells, 7,160,000; white cells, 6120; hæmoglobin, 136 per cent (Sahli); and hæmatocrit readings, 65 and 75. A diagnosis of congenital pulmonary atresia was made by Dr. William Evans who noted the limited cardiac signs despite the great cyanosis.

Dental extractions were performed uneventfully under gas and oxygen anæsthesia. Venesection on three occasions had no effect upon the patient's condition or blood picture. She was discharged on March 14. On April 2 she was readmitted after an epileptiform fit preceded by difficulty in speech and loss of power in the left side of the face and left arm. The general appearance was unaltered unless the cyanosis was deeper, and there was now orthopnœa.

The temperature was 102 and the pulse rate 88. The cardiac signs were unchanged. There was drowsiness, slurring of speech, and neck rigidity. Slight left facial weakness of upper motor neurone type was combined with flaccid weakness of the left arm with absent tendon reflexes. These were the only abnormal signs in the nervous system. After admission the patient had several minor, and one major, epileptiform attacks. Lumbar puncture showed a yellow C.S.F. under pressure of 230 mm. It contained red cells (800 per

FIG. 1.—Photograph of patient showing cyanosis and clubbing of fingers.

c. mm.), no excess of white cells, and protein 30 mg. per 100 c.c. In subsequent days she improved, the nervous symptoms and signs disappeared, and she was fit to get up. But one month after admission she developed a sudden pain under the left costal margin with fever, attributed after necropsy to a paradoxical splenic infarct. The spleen was never felt, but great œdema of both lower limbs with tenderness in the groins indicated spreading iliac thrombosis. The general condition deteriorated and she died in coma on May 13, 1942, eight days after the final thrombotic illness.

SUMMARY OF NECROPSY (DR. W. W. WOODS)

*Heart failure. Thrombosis in cerebral and pelvic veins. Congenital atresia of orifice of pulmonary artery. Patency of interventricular septum. Enlargement of bronchial arteries.*

Small pulmonary artery (1·3 cm. long; 1·3 cm. circumf.) starting from a blind end at upper margin of right ventricle and dividing into very thin-walled right and left pulmonary arteries (each 1·8 cm. circumf., with wall less than 0·05 cm. thick), which go into hila of lungs. No trace of conus arteriosus or pulmonary valves on inner surface of right ventricle (Fig. 3). Patency (3 cm. diam.) of upper end of interventri-

FIG. 2.—Teleradiogram, showing *Cœur en Sabot* and deep pulmonary bay with small hilar vessels.

cular septum immediately below anterior cusp of aortic valve and not involving pars membranacea septi. Greatly enlarged, very thin, almost transparent, right bronchial artery (2·5 cm. circumf.) arising (orifice 1 cm. diam.) from the aorta immediately beneath the level of the closed ductus arteriosus (Fig. 4), its three branches going to the three lobes of right lung. Upper left bronchial artery (0·5 cm. circumf.) arising from aorta, 0·7 cm. below the right, by an incongruously wide orifice (1 cm. diam.) and entering hilum of left lung. Lower left bronchial artery (1·2 cm. circumf.), with thin, almost transparent wall, arising (orifice 0·5 cm. diam.) from aorta 0·7 cm. below upper, and entering lower part of hilum of left lung. Foramen ovale and ductus arteriosus closed. Great hypertrophy of right ventricle (2 cm. thick), forming apex of heart. Small left ventricle (1 cm. thick). Normal pulmonary veins entering left auricle. Aortic orifice 9·5 cm. in circumference. Three abnormally large aortic cusps (width, upper edge, 4 cm.; depth, 2 cm.). Slight fibrous thickening of free margin of tricuspid valve and of free margins of aortic cusps. Large coronary arteries (right 1 cm., anterior descending 1·5 cm. circumf.). Slight atheroma of aorta. No atheroma of

FIG. 3.—Right ventricle opened, showing:

(A) Ventricular septal defect.
(B) Hypertrophied wall of right ventricle.
(C) Site of pulmonary atresia.
(D) Tricuspid valve leading to right auricle.

FIG. 4.—Aorta opened, showing:

(A) Dilated orifices of (upper) one right and (lower) two left bronchial arteries, maintaining pulmonary circulation.
(B) Normal intercostal arteries.
(C) Aortic valve.

bronchial arteries. Emphysema and moderate œdema of lungs. Very great dilatation of large left (1·2 cm. diam.) and right (1·7 cm. diam.) superior anastomotic vein of Trollard, posterior anastomotic vein of Labbé, and superficial Sylvian vein. These veins on right and a few tributaries of the right vein of Trollard filled with firm ante-mortem thrombus (Fig. 5). Canalized occlusion of termination of left superficial Sylvian vein. Cavernous and circular sinuses unfortunately not carefully examined during removal of pituitary body. No thrombosis nor dilatation of superior longitudinal and lateral sinuses. No petechiæ nor softening in horizontal sections of right cerebrum. Considerably adherent ante-mortem thrombus filling right common iliac vein and upper end of right external iliac. Less adherent ante-mortem thrombus filling left common iliac vein. Non-adherent ante-mortem thrombus filling lower fourth of inferior vena cava. More recent ante-mortem thrombus in femoral veins. Moderate œdema of left leg; slight of right. Recent ante-mortem thrombus in very dilated veins (0·6 cm. diam.) in myometrium of fundus of uterus, in parts of very conspicuous plexus of veins in each broad ligament, throughout dilated right ovarian vein and in lower end of dilated left ovarian vein. Bicornuate uterus. Softened anæmic infarct (7×3 cm.)

in œdematous, congested spleen, presumably due to an embolus from a thrombosed vein having crossed through the patent interventricular septum. Lightly adherent thrombus riding bifurcation of aorta, probably also a crossed embolus. Congestion and nephrosis of large, lipoid-flecked, kidneys. Diffuse congestion of liver, stomach, pancreas, and bladder.

*Microscopical Examination*

The brain and portions of kidney and lung were placed in 4 per cent saline formaldehyde. In the time available it was possible only to stain frozen sections by Herxheimer's method and to embed tissue in paraffin by a rapid method, using the exhaust pump, and to stain with hæmatoxylin and eosin, Weigert's iron hæmatoxylin with van Gieson, Weigert's fuchselin with neutral red, and by Lenhossek's method for Nissl bodies.

*Brain.* A horizontal segment was taken from the right cerebrum, where above the sharp anterior bend of the vein of Trollard a piece of the post-central gyrus is exposed (Fig. 5). The segment included the thrombosed vein, the post-central gyrus, the

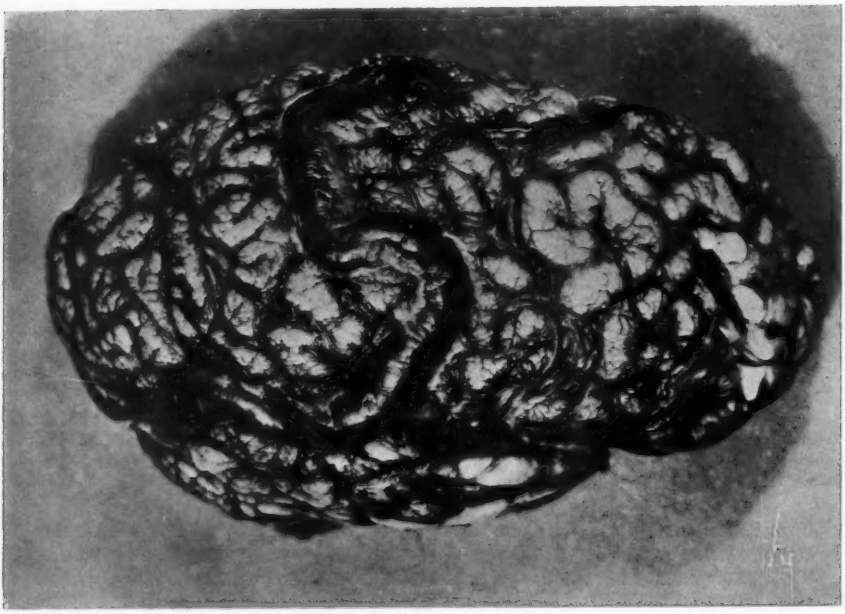

FIG. 5.—Right hemisphere; enlarged, thrombosed superior anastomotic vein of Trollard and posterior anastomotic vein of Labbé, joining superficial Sylvian vein. The anterior end is to the right, and the brain has been tilted so that the anterior and upper parts are more prominent.

inferior post-central sulcus, and the pre-central gyrus near the junction of its upper and middle thirds. Two other segments were taken from the inferior frontal gyrus, 3 and 3·5 cm. from the anterior pole, one for Herxheimer's method and one for rapid paraffin embedding.

The clot in the vein of Trollard is red, with a few small mixed areas. In its outer margin are scattered pigment granule cells, while early organization is shown by capillaries and a few fibrocytes. There is partial thrombosis of a vein deep in the

inferior post-central sulcus. The pia-arachnoid over the vein of Trollard and extending over the greater part of the summit of the post-central gyrus is greatly infiltrated with free, round endothelial cells; some contain pigment. In a narrow zone immediately above the vein are numerous neutrophil leucocytes. The pia in the inferior post-central sulcus is fibrosed, containing numerous fibrocytes, and is infiltrated chiefly with lymphocytes but also with monocytes, epithelioid cells, and a few neutrophil leucocytes and pigment granule-cells. Where the centre of the convexity of the vein is opposite the post-central gyrus the adjacent cortex, including the layer of small pyramidal cells, is densely infiltrated with rod and other apparently microglial cells. Similar cells extend in decreasing number to the polymorphic layer. In this anterior part of the gyrus, and to a less degree in the posterior, there is an obvious excess of small round, apparently oligodendroglial, cells about neurons, especially in the deeper layers. In the pre-central gyrus there is conspicuously less cellularity but the satellite cells in the deeper layers appear to be in excess of those in the prefrontal section. Even in the post-central gyrus few of the pyramidal cells have lost their shape and in the pre-central gyrus they are well shaped and do not differ from those in the prefrontal region. The nuclei of neurons are central, though the nucleoli are occasionally peripheral. The Nissl bodies are almost always finely granular and indistinct, though larger fused masses are occasionally seen in the periphery of the cytoplasm and most of the Betz cells contain deeply stained rounded masses. In the prefrontal section, however, Nissl bodies show similar changes, so that their condition everywhere is probably due to post-mortem change or rapid fixation and embedding. The conspicuous perivascular, perineuronal, and pericellular spaces of His demonstrate faulty fixation or embedding. There are no hæmorrhages. There are no fat granule-cells in the Herxheimer sections, which do not, however, include the thrombosed vein and the post-central cortex immediately to its side.

*Kidneys*. Greatly congested; glomeruli, large with considerable proliferative glomerulitis. Three glomerular adhesions were found, and there is early fibrosis and infiltration with lymphocytes and an occasional neutrophil leucocyte round a few glomeruli or about their afferent vessels. In the interstitial tissue a few spindle cells and small groups of foam cells contain lipoid; lipoid is present less often in cells of first convoluted tubules.

*Lung, right lower lobe*. Congested and emphysematous; emphysema severe and chronic, and alternating with scattered patches of partial collapse, in which the alveoli usually contain a coagulated exudate. No bronchiolitis. Pulmonary arteries well developed.

## Discussion

Pulmonary atresia is an uncommon defect, Abbott (1936) finding 40 examples in 1000 confirmed cases of congenital morbus cordis. We have examined the necropsy reports of the London Hospital, covering a period of 31 years, and found 355 cases of congenital morbus cordis, other than those showing patent foramen ovale alone; among these were 16 cases of pulmonary atresia. Two main types are described, those in which the interventricular septum is closed and the pulmonary circulation is maintained by way of a patent foramen ovale, a dextroposed aorta, and a patent ductus arteriosus; and those in which the interventricular septum is perforate. The prognosis is different, being far worse in the first type with an average duration of life of 3 months and a maximum of 6 months, against an average of 3·4 years with a maximum of 13 years in the second type, as given in Abbott's (1928) series of 31 cases. Harrison (1929)

comments on the relatively favourable prognosis where the interventricular septum is patent, and cases, aged 33 and 30, have been reported by East and Barnard (1938), and Bach (1928), respectively. The collateral circulation in this type has been fully discussed by Harrison (1929), who emphasized the importance of the bronchial arteries in maintaining it when the ductus arteriosus was closed, and gave references. This was recently noted in two cases by East and Barnard (1938), and it was a feature in our case, where incidentally the aorta sprang normally from the left ventricle and was not dextroposed.

The bronchial arteries normally arise from the aorta or intercostal arteries and supply the lung tissue (Gray's Anatomy, 26th edition, 1935). They vary in number from one to three for each lung, generally with two on the left and one on the right, and with an anastomosis between these vessels and the branches of the pulmonary artery. The compensatory circulation may also be maintained by a patent ductus arteriosus, by the mediastinal, œsophageal, and other branches of the aorta, or by anomalous vessels arising from the subclavian and thyroidea ima arteries (Abbott, 1932).

The diagnosis of pulmonary atresia cannot always be made during life as the syndrome of gross cyanosis and clubbing of the fingers, with the *cœur en sabot* of right ventricular enlargement resembles closely the commoner form of Fallot's tetralogy where there is stenosis, not atresia. If a harsh systolic murmur and thrill in the pulmonary area are added the distinction seems impossible. But sometimes, where the septal defect is large, as in our case, the murmur is slight and the thrill absent; this should suggest the diagnosis.

Cerebral manifestations in pulmonary atresia and stenosis are well recognized. They have been attributed either to paradoxical embolism, or to cerebral thrombosis associated with the polycythæmia. We here record hemiplegia and epileptiform attacks which were shown at necropsy to be due to cerebral thrombosis. Their transient nature may be explained by the absence of softening of the brain although there was such extreme distension and thrombosis of the cerebral veins (see Fig. 5).

We are glad to acknowledge the kindness of Professor H. M. Turnbull, F.R.S., and Dr. W. W. Woods of the Bernhard Baron Institute of Pathology in furnishing us with the detailed necropsy report, and with Fig. 5. Professor Turnbull has kindly permitted reference to past records.

Dr. A. E. Clark Kennedy, Physician to the London Hospital, was good enough to allow us to publish this case which was admitted under his care, and Dr. William Evans has helped us in the Cardiac Department.

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# PARTIAL HEART BLOCK DUE TO DIGITALIS

BY

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Amongst a series of cases of partial heart block, collected over a period of years, there were some in which treatment with digitalis was mainly or partly responsible. This group involves some special problems that seem worth separate consideration.

The slow pulse, even as slow as 35 in a minute, after the administration of digitalis was one of the original observations of Withering (1785). Much later, but for many years now, it has been recognized that heart block was the cause of the slowest heart rates after digitalis, though some lesser degree of slowing might be due to a direct or vagal effect on the S-A node. According to Cushny (1925, p. 220) the effect of digitalis on the A-V bundle is partly inhibitory through the vagus nerve, but partly, and sometimes mainly with full or long digitalization, a direct effect on the muscle of the A-V bundle.

The slow pulse after digitalis has even passed into literature and, if Mary Webb can be trusted, into the folk-lore of Shropshire and the Welsh border:

> "I suppose a dose of foxglove ud put her right, maybe?"
> "Ah! Foxglove'll lower the pulse as quick as anything. But you mun be careful. Dunna give her too much. When things get old and worn out they canna stand much of it." (Mary Webb (1924), Precious Bane.)

Mackenzie (1905, *b*) pointed out that those who already had partial heart block, as shown by a long a-c interval, were more susceptible to this effect of digitalis and were more likely to develop a recognizable degree of heart block. This is confirmed by the present study.

This series was obtained by looking through 10,000 consecutive electrocardiograms taken at Guy's Hospital during the twelve years 1927–38, and adding all others I had remembered to index. There were 141 cases with a prolonged P–R interval but no higher degree of block; and the term latent is used as a convenient one to describe this, since it is a true subdivision of heart block that is only made manifest by instrumental methods. There were 29 which also had, at times, partial heart block with dropped beats.

In 26 of these 170, treatment with digitalis was the sole or an important cause of the heart block, and in another 6 it may have been the cause. However, these digitalis cases were not equally distributed: as many as 38 per cent of those with dropped beats were due to digitalis or similar drugs (Table I), but

only 14 per cent of those with latent heart block (26 per cent of those with a P–R interval of 0·26 sec. or more, and 12 per cent of those with it between 0·25 and 0·20 sec.)

TABLE I

HEART BLOCK CAUSED BY OR INCREASED BY DIGITALIS

| | Total number of cases | Number due to or increased by digitalis (*a*) | Number possibly due to digitalis (*b*) | Percenta due to digitalis (*a*) and (*b*) |
|---|---|---|---|---|
| Partial heart block with dropped beats .. .. .. .. | 29 | 11 | 0 | 38 |
| Latent heart block— | | | | |
| (*a*) P–R, 0·26 and above .. | 31 | 7 | 1 | 26 |
| (*b*) P–R, 0·20–0·25 sec. .. | 110 | 8 | 5 | 12 |
| | 170 | 26 | 6 | 19 |

To write of digitalis without discussing auricular fibrillation is of course to leave out its most important use. The high degree of heart block that is often produced is well known; but here we are not concerned with this aspect of digitalis overdosage.

## CASES WITH DROPPED BEATS

The eleven cases with dropped beats are summarized in Table II, and short notes of two of them follow to give more illustrative details.

*Case* 2. A man, aged 24, with a large heart and mitral stenosis, was admitted to hospital for his second attack of congestive failure. His P–R interval at various times without digitalis was 0·23–0·25 sec. He was given 30 minims of tincture of digitalis daily from May 8 to 31, when block was noted, and four days later there was still 2:1 heart block, partial heart block with dropped beats, and sino-auricular block (Fig. 1). Many records were obtained and the average results are shown below.

2:1 heart block: P–R, 0·25 (almost his normal), dropped beat.
3:2 block: P–R, 0·24, 0·33, d.b.

Single records of lesser degrees of block are shown below.

4:3 block: P–R, 0·25, 0·31, 0·39, d.b.
5:4 block: P–R, 0·25, 0·31, 0·36, 0·39, d.b.
6:5 block: P–R, 0·26, 0·31, 0·35, 0·39, 0·42, d.b.
7:6 block: P–R, 0·25, 0·29, 0·32, 0·36, 0·39, 0·42, d.b.

On May 24 his P–R interval had been 0·25 sec., and on May 26 and May 28 his temperature, which had been normal, rose to 100·6° F., suggesting that a return of active carditis was the decisive factor in bringing about his heart block on May 31. His recovery was uneventful and twelve days later the P–R interval had returned to 0·25 sec.

*Case* 4. A woman, aged 68, had congestive heart failure and high blood pressure. Treated with 45 minims of tincture of digitalis daily, her pulse rate, which had been 100–80 on admission, was charted at under 50 on the tenth day, and digitalis was stopped. Dropped beats were recorded, generally with the sequence: P–R, 0·23 sec., 0·31 sec., d.b., etc. The heart rate remained about 50 for ten days, and that day

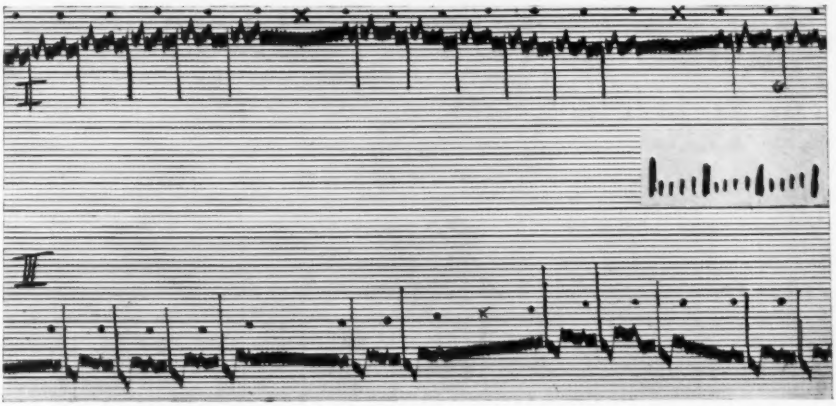

FIG. 1.—Dropped beats after 2, 3, 4, 5, and 6 responses with a lengthening P–R interval. The auricular waves are marked with a dot (·); in three of the longer pauses (marked with a cross) and perhaps in a fourth there is also sinoauricular block. The auricular rate was about 60. In some other records 2:1 heart block was the most usual finding. The time marker is not visible in this print and has been marked on in seconds and fifths. (Case 2).

dropped beats were again recorded with the average sequence of P–R, 0·21 sec., 0·41 sec., d.b., etc. After another seven days, during which the heart rate was regularly over 80, she was started again on digitalis—this time 2 grains of the leaf daily—and took this regularly, the P–R interval remaining just below 0·21 sec., as at first. It is strange that so little digitalis should have produced this and probably infection was the extra cause (see page 135).

### DISCUSSION OF CASES WITH DROPPED BEATS

All these eleven patients had dropped beats, developing as the first sign of over-digitalization or quickly after this, and disappearing within a few days when it was stopped. Sometimes the dropped beats were only observed for a day or two so that we did not obtain a cardiogram, the patients being under the care of various physicians in different wards. Most often the dropped beats persisted, for from three to five days, and in one case at least they were present the greater part of the time for ten days.

*The P–R Interval before Digitalization.* In nine cases digitalis was being used in some form; in one, strophanthin; and in one (Case 28), quinidine; the last has been described elsewhere and is only mentioned as evidence that quinidine can produce the same effect (Campbell, 1932). In seven of the remaining ten, the P–R interval without digitalis was known and the figures were 0·16, 0·20, 0·21, 0·21, 0·22, 0·24, and 0·25 sec. Thus it was prolonged in all except the first (Case 27) where an unreasonable amount of strophanthin had been given to a patient with pyrexia because of tachycardia after tonsillectomy. This bears out the experience of Mackenzie (1905, *b*) that it is the patient with latent heart block who is apt to develop dropped beats under digitalis treatment.

Apart from the presence of latent heart block there seemed no other ætiological factor common to all these cases. Rheumatic and syphilitic valvular

disease and myocardial disease, with and without high blood pressure, were all represented. Probably rheumatic disease formed a smaller proportion than in many conditions because of the tendency of rheumatic hearts to fail with auricular fibrillation, which would automatically exclude them from this study. The effect of digitalis on the P–R interval in these patients is shown in Table II.

TABLE II

THE PRODUCTION OF DROPPED BEATS FROM DIGITALIS THERAPY

| Case No. | Sex and age | Diagnosis | P–R interval | | | Digitalis treatment | |
|---|---|---|---|---|---|---|---|
| | | | with digitalis | | without digitalis | | |
| | | | with dropped beats | without dropped beats | | Daily amount | No. of days |
| 2 | m. 24 | Mitral stenosis; C.F.*; active carditis. | 0·24–0·42 | — | 0·24 | 30 minims of tincture | 23 |
| 4 | f. 68 | High blood pressure; C.F.*; pyrexia. | 0·23–0·31<br>0·21–0·41 | — | 0·21 | 45 minims of tincture | 10 |
| 8 | m. 44 | High blood pressure; C.F.*; pyrexia. | 0·25–0·29 | 0·25 | — | 3 grains of leaf and later 20 minims | 18 |
| 9 | f. 66 | Left ventricular failure (died). | 0·24–0·42 | 0·40 | — | 6 grains of leaf | 5 |
| 11 | f. 41 | High blood pressure; C.F.*; (died). | 0·18–0·26 | 0·26 | 0·20 | 45 minims of tincture | 14 |
| 12 | f. 72 | Carcinoma; C.F.* | — | 0·28 | — | 69 minims of tincture | 9 |
| 18 | f. 36 | Mitral stenosis; acute rheumatism. | 0·23–0·38 | 0·30 | 0·25 | 1/80 grain of Nat. dig., and quinidine | ?6 |
| 19 | f. 58 | High blood pressure; C.F.* | 0·19–0·38 | — | 0·21 | 60 minims of tincture | 14 |
| 26 | m. 41 | Syphilitic A.I.; C.F.*; pyrexia. | — | 0·29 | 0·22 | 60 minims of tincture | 33 |
| 27 | f. 31 | Tonsillectomy; pyrexia | — | 0·28 | 0·16 | 3/200 grain of strophanthin | 7 |
| 28 | m. 40 | Thyroidectomy for hyperthyroidism | 0·23–0·46 | — | 0·18 | 15 grains quinidine | — |

* C.F.=congestive failure.

*Infection or Pyrexia.* Although latent heart block was already present in nearly all these cases, and in addition in most of them congestive failure, yet another conditional cause was generally present. Of the ten, two had active rheumatic carditis (one with failure) and four others (three with failure) had some degree of pyrexia, at the time, so that there was probably some infection making the heart more susceptible to digitalis.

In the first two with active rheumatic carditis (Cases 2 and 18) it is not surprising that dropped beats developed as they are liable to occur in this condition, even without digitalis. In a third (Case 27) there had been a fairly severe infection with pyrexia for some time. In her case, however, the amount of strophanthin given was excessive, and in Case 18 also the amount of digitalis was more than is usual and certainly excessive when quinidine was given at the same time.

Relatively slight infections seem to have had an important effect on the heart in the other three. In one (Case 26) there was "influenza" with a temperature for three days and probably it would hardly have been referred to in the notes if heart block had not developed. The sequence of events is illustrated in Fig. 2. Digitalis was stopped after he had taken 60 minims of the tincture daily for 33 days because the heart rate fell to an irregular 50 a minute (seventh day of fig. 2). For four days it varied between 50 and 80, and one day the P–R interval was 0·29 sec. though no dropped beats were recorded. The heart rate, then, remained about 80; and after seven days' break digitalis

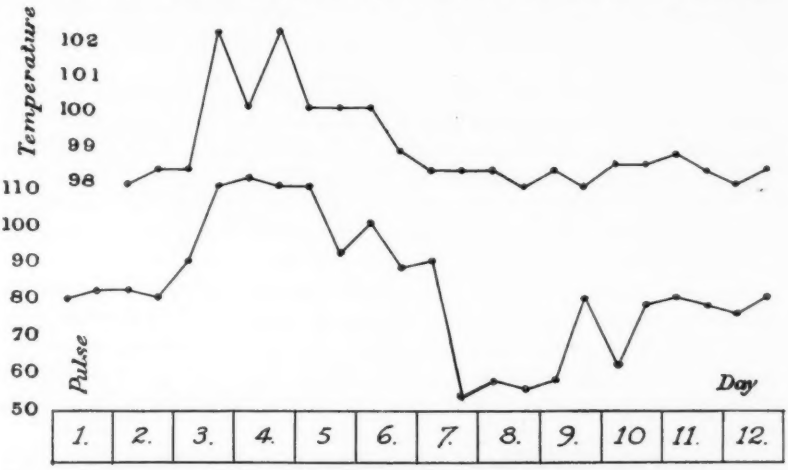


FIG. 2.—A slow pulse due to heart block after a transient infection. The upper line represents the temperature which returned to normal after four days (third to sixth days inclusive). The lower line represents the heart rate which rose with the onset of the temperature but fell sharply five days later (seventh day in fig.) when heart block developed. (Case 26)

was resumed, this time 45 minims daily, and continued until his discharge four weeks later, without any return of dropped beats. On admission with failure and without digitalis, the P–R interval had been 0·22; on discharge with digitalis (45 minims daily) but without failure, it was 0·23; and six months before without either, it had been 0·18 sec.

In the other two the pyrexia was even more trivial and no special cause for it was found. However, in one (Case 8) there was little doubt of the connection because the first time dropped beats were observed there had been a rise of temperature to 99·5° or 100° each evening for eight days; and the second time the dropped beats occurred when the rise was about its maximum, reaching 100° F. every evening; after this there was no more pyrexia, and for the remaining three weeks while he was in hospital he was able to continue with the same amount of digitalis without any recurrence of dropped beats. In the other (Case 4) the connection again seemed a close one, but not proved so clearly, as her treatment was resumed with a smaller dose of digitalis. Two days after her block was first observed her temperature rose to 100° F. for two days, and again

for three days at the time the second cardiogram showing block was taken, when she had already been without digitalis for ten days.

In none of the last three cases was there any reason to think that the slight pyrexia was a sign of recurrent, but undiagnosed, rheumatic carditis; one had syphilitic aortic incompetence with congestive failure, and the last two were elderly patients with congestive failure secondary to high blood pressure.

Cushny (1925, p. 90) states that as the temperature is raised experimentally the heart, whether isolated or in the intact animal, becomes more susceptible to digitalis; this being due to a direct action on the muscle as well as to the effect of the acceleration of the heart rate. It seems unlikely, however, that these small rises of temperature in transient infections would be enough to act in this way.

Thus six of the ten cases had some infection at the time of their dropped beats. Two others had hearts that were very gravely damaged as shown by the fact that one (Case 9) died suddenly the next day (see p. 140) and another (Case 11) died three weeks later in spite of all treatment. Cushny (1925, p. 225) says that when the heart is reduced to a state of malnutrition the block through direct muscular action is attained early and easily from digitalis, so that this may have been an additional cause in these two cases.

It seems, therefore, that the presence of infection or of a heart that is almost moribund makes a patient prone to develop partial heart block with dropped beats from an amount of digitalis that would not ordinarily produce this. Three cases supported this directly by being able to take the same amount of digitalis, when they had no pyrexia, without dropped beats developing (Cases 2, 8, and 26).

Accepting this, there were only two patients (Cases 12 and 19) who developed dropped beats under treatment without any additional cause: both had been given 60 minims of tincture of digitalis daily, one for 14 days and the other for 9 days. Perhaps 14 days may be rather longer than many patients can take this amount, but another (Case 26) had taken it for 33 days and even then the onset of an infection was certainly a partial cause of his dropped beats. The quantity of digitalis that was being taken by all these cases is discussed later (see p. 139).

*The Effect of Digitalis on the P–R Interval.* This is shown in Table II. The average P–R interval before digitalis therapy was 0·21 sec. In seven cases there was a record of the P–R interval about the time of the dropped beats, but at a moment when they were absent and there was latent heart block without dropped beats. The average figure was 0·29 sec.

Where there were dropped beats they generally occurred after two normal, if slow, responses, i.e. 3:2 heart block (and this was the only group where there were enough for statistical treatment). In three the P–R interval of the response before the dropped beat was moderately prolonged, the average figure being 0·22 sec., 0·29 sec., dropped beat. In the other six the P–R interval of the response before the dropped beat was much longer, and the average figures were 0·22 sec., 0·41 sec., dropped beat. It did not seem possible to find any reason for the difference between these two groups; in fact one patient (Case 4) after

an interval of five days varied just as much as these two very different sets of figures.

The P–R interval at a time when there were no dropped beats was on the average about half-way between the two P–R intervals that came before the dropped beat; but this was only an average of very divergent figures, and in individual cases it might be little longer than the first P–R interval or nearly as long as the second.

The first P–R interval after the dropped beat was on the average much the same as the P–R interval without any digitalis, and here there was much closer agreement in individual cases.

### Latent Heart Block without Dropped Beats

In addition to the 11 cases with dropped beats, there were 15 others where digitalis treatment lengthened the conduction time, and another 6 (making 21 in all) where this was probable, but not quite certain, as there was no record of the usual P–R interval without digitalis. In 8 of these the 21, P–R interval was 0·26 sec. or more; some details of these cases are shown in Table III.

TABLE III

Prolongation of the P–R Interval to 0·26 sec. and above by Digitalis

| Case No. | Sex and age | Diagnosis | P–R interval | | Daily amount of digitalis |
|---|---|---|---|---|---|
| | | | with digitalis | without digitalis | |
| 45 | f. 15 | Mitral stenosis .. .. | 0·36<br>0·40 | 0·28*<br>0·28* | 45 minims tincture<br>3 grains leaf |
| 48 | f. 18 | Acute rheumatism; aortic incompetence. | 0·31 | 0·20 | 5 gr. of leaf for several days |
| 57 | m. 54 | Mitral stenosis; congestive failure. | 0·34 | 0·26† | 1/100 gr. Nat. dig. for several days |
| 41 | m. 68 | High blood pressure; auricular flutter. | 0·30 | 0·24 | 1/150 gr. Nat. dig. for 7 days (see text) |
| 61 | m. 51 | Left ventricular failure .. | 0·28 | 0·18 | 75 minims tincture for 21 days |
| 38 | m. 58 | High blood pressure; congestive failure. | 0·28 | 0·20 | 80 minims tincture for 28 days |
| 53 | f. 47 | High blood pressure; congestive failure. | 0·27 | — | 45 minims daily |
| 46 | m. 60 | Heart failure; coronary thrombosis. | 0·26 | 0·21 | 1/200 gr. Nat. dig. for 18 days |

* Some years later it was 0·36 without digitalis. † Still on some digitalis.

*The P–R Interval before Digitalization.* Some degree of heart block before digitalis treatment seemed the most important factor in allowing digitalis to increase this significantly. Where the P–R interval was increased to 0·26 sec. or above by digitalis, only one of the seven where the P–R interval was known had one of less than 0·20 sec. without digitalis; his was 0·18 sec. and he was given 75 minims of the tincture daily for 21 days (Case 61). In the eighth the P–R

interval without digitalis was not known. The average figures for the P–R intervals before and after digitalis were 0·22 and 0·30 sec. respectively.

Where the P–R interval was increased to something less than 0·26 sec. by digitalis, it was above 0·18 sec. without digitalis in all of the eight, except in one with acute infection and severe congestive failure and in one during pregnancy; in both these it was only 0·16 sec. There were five others in this group where the P–R interval without digitalis was not known. The average figures before and after digitalis were 0·19 and 0·22 sec. respectively.

*Other Aetiological Factors.* The presence of latent heart block before digitalization was the main factor that was common to nearly all of the cases. The type of heart disease seemed of no significance and all varieties were present in different patients; nine of the twenty-one had valvular disease, nearly always rheumatic, and the other twelve had primary myocardial disease, six of these with high blood pressure and three at least with disease of the coronary arteries.

Heart failure was naturally present in the great majority as it is the main indication for digitalis. The reasons for its administration in four of the other six without failure was tachycardia and cyanosis (Case 45), some undue dyspnœa during pregnancy, auricular flutter (Case 41), and frequent extrasystoles; in the other two there seemed to have been no adequate reason for the prescription of digitalis.

The other factor that seemed common to these cases—at any rate to those where the P–R interval was most increased—was a rather bad prognosis. There were eight in whom it was lengthened to 0·26 sec. or more, and two of these were not followed for long enough to know the outlook. Of the other six, one did well, one had acute pulmonary œdema in the fifth year, and four died after 1, 1, 4, and 8 years respectively. As digitalis was generally used for a failing heart, and heart failure with normal rhythm has a worse prognosis than failure with auricular fibrillation, one could not expect the patients to live for very long, but these figures are worse than might be expected.

*Infection or Pyrexia.* Active infection was much less in evidence as a cause than in the cases with dropped beats, but was present in three. One had infective endocarditis and congestive failure. A second, a girl of 18, to whom 5 grains of digitalis leaf has been given daily for several days without any adequate reason had a recurrence of acute rheumatism at the time (Case 48; P–R from 0·20 to 0·31 sec.). A third, a boy of 7, was critically ill with congestive failure due to acute myocarditis and acute nephritis following tonsillitis: probably, he owed his life to digitalis treatment, though the P–R interval lengthened from 0·16 to 0·21 sec.; this last may partly have been due to his myocarditis, as some of the inversion of the T waves certainly was, but seemed more closely related to the time of his treatment with digitalis than of his active infection. A fourth was recovering from recent broncho-pneumonia, but this seemed rather the exciting cause of his congestive failure than of his partial heart block (Case 57).

One case where the P–R interval was not lengthened by digitalis was striking in this connection. A woman, aged 56, suffering from the after effects of tonsillitis and broncho-pneumonia some months before and from auricular

fibrillation that was probably due to old hyperthyroidism, had a P–R interval of 0·13 sec. after treatment with 45 minims of tincture of digitalis daily for five weeks; fibrillation had stopped after a few days treatment with digitalis and may possibly have been paroxysmal. Four months later she was again admitted with an acute rheumatic infection, most unlike rheumatic fever and with no other signs of cardiac involvement, and while she was being treated with sodium salicylate only, her P–R interval was 0·22 sec.; presumably this was due to the infection, and it had been only 0·13 sec. when she was treated with digitalis.

## DIGITALIS

*The Amount of Digitalis.* In few of these patients could the increase in the heart block be attributed to an amount of digitalis that was obviously excessive, and curiously enough the largest amounts had sometimes been given where there seemed no indication for digitalis therapy (Cases 18, 27, and 48).

In the ten where dropped beats developed, three had large doses of digitalis—1/240 grain of Nativelle's digitaline, t.i.d., and some quinidine, probably for 6 days (Case 18, with acute rheumatism); 1/200 grain of strophanthin, t.i.d., for 7 days (Case 27, with tachycardia and pyrexia after tonsillectomy); and 6 grains of the leaf daily for 5 days (Case 9, with left ventricular failure). Certainly in the first two there seemed no reason why digitalis should have been given. Three others had 60 minims of tincture daily for 9, 14, and 33 days, and even then the last only developed dropped beats with an intercurrent infection (Case 26). Of the remaining four, two had the standard dose of 45 minims daily and two had only 30 and 20 minims daily. In the last two these amounts had been taken for 23 and 18 days respectively; and recurrent infection (once rheumatic and once of unknown origin) was almost certainly the precipitating cause, as later when they had no pyrexia they were able to take the same amount without the production of dropped beats.

In the group of eight, with the greater increase of conduction time but no dropped beats, four had amounts that were large—5 grains of the leaf daily for no apparent reason (Case 48, with recurrent rheumatism); 1/100 grain of Nativelle's digitaline daily for several days (Case 57); 75 minims of the tincture daily for 21 days (Case 61); and 80 minims of the tincture daily for 28 days (Case 38); these two last, however, were both large heavy men.

In the other four of these eight, and in the thirteen with the lesser increase in the conduction time, the amount of digitalis was certainly not unusually large. In two only, who had about 60 minims daily, was it above 45 minims of the tincture daily (or its equivalent), and in five it was less than this.

The cause, therefore, rarely lay in excessive amounts of digitalis, and this might be expected where most cases were being treated at hospital, though it is only fair to add that during the years in question massive digitalization has not been a popular method of treatment at Guy's Hospital—and, in my opinion, rightly so. In general, during continued treatment, failing to give an adequate amount of digitalis is much more common than giving too much.

*Other Symptoms.* Little has been said about other symptoms produced by

the overdose with digitalis. Price (1941) states that they are anorexia, nausea, vomiting, and diarrhœa, in addition the very varied effects on the rhythm. Unfortunately in this series of cases special notes were not taken about the general condition of the patient, and no useful conclusions can be drawn about the relative stages at which the general symptoms and the slower pulse occurred. Nausea and vomiting were noted most, but it is surprising how often there was no note of other symptoms, and how the house officer in charge did not stop the exhibition of digitalis until partial heart block with dropped beats was observed. It is also surprising that extrasystoles were not noted; no single case was observed where they were seen in the cardiographic record at the time of full digitalization, though of course they would have been difficult, if not impossible, to recognize clinically when there were dropped beats.

*Indications for Digitalis in Heart Block.* The old contraindications to digitalis therapy have gradually been swept away, and Cushny (1925, p. 233) in lending his authority to this view agrees that digitalis may be useful even when there is complete heart block, but still looks with doubt at its use when there is partial block. Obviously the risk of increasing this must be taken into account; but when failure is present, digitalis should always be used in adequate amounts until experience shows that the drawbacks, if any, outweigh the advantages.

These results have made it clear that when there is congestive failure with latent heart block, an increase in this, even with dropped beats, is liable to occur after reasonable amounts of digitalis. There seemed, however, no great drawback even in cases where dropped beats were produced, and in general the block passed off quickly within two to four days of the digitalis being stopped, though once it persisted for ten days. As might be expected from its slow absorption and slow effect, there was sometimes a higher degree of block the day after digitalis had been stopped, and this would be a danger if the condition was not recognized. Freedom from ill-effects is dependent, therefore, on the heart block being recognized with reasonable speed, but care and knowledge are always needed to make the most effective use of digitalis.

One of these patients died suddenly, soon after block had been observed and digitalis stopped; but she was extremely ill before and had, post-mortem, advanced disease of the coronary arteries with a badly scarred ventricle, so that sudden death was an ever-present risk; it is possible but far from certain that digitalis may have caused her death by producing ventricular fibrillation; it is probable that it had already prolonged her life in previous attacks.

When failure is not present, there are fewer indications for digitalis and the recognition of partial heart block may decide against it. This, however, must not be made a general rule, and one case is of special interest because the high degree of block might have prevented treatment with digitalis in a girl who was greatly benefited and continued taking it most of the time for several years. She had high grade mitral stenosis with much cyanosis and dyspnœa. Dozens of electrocardiograms between 1931 and 1938 never showed a shorter P–R interval than 0·26 sec.; in 1932, 45 minims of the tincture daily increased it from 0·28 to 0·36 sec., and in 1933 a period of treatment with 3 grains of the leaf daily increased it to 0·40 sec. (see Fig. 3). Gradually, during the course

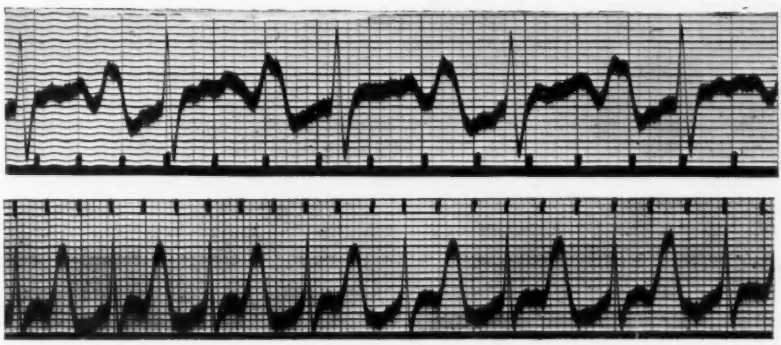

FIG. 3.—Latent heart block, present for eight years without a dropped beat being noted,
(A) Above. 13/5/31. The shortest P–R interval of 0·28 sec.
(B) Below. 24/11/33. The P–R interval had been increased from 0·28 to 0·40 sec. by digitalis, but later it increased to 0·36 sec. without digitalis.
The P wave was always large, but in (B) it is exaggerated as the tachycardia and the still longer P–R interval have superimposed it on the T wave. (Case 45)

of these seven years, the P–R interval rose from 0·26 to 0·36 sec., even when she was not taking digitalis. It is surprising that at no time (and she attended hospital regularly for years) was a dropped beat observed (Case 45).

Mackenzie (1905, *a*) described a somewhat similar case where the a–c interval was two-fifths of a second and remained so for twelve years; no dropped beats were observed except for one short period about the middle of the time. He emphasized that digitalis must be used whenever indicated, even in partial heart block, and that sometimes when there was effective slowing the conductivity might even be improved. Cohn and Fraser (1913) have suggested, as an explanation of how digitalis may help in some cases of mitral disease, that it may act by lengthening the A-V interval and thus facilitating the passage of blood from auricle to ventricle.

## SUMMARY AND CONCLUSIONS

Digitalis therapy is one of the commonest causes of partial heart block with dropped beats, and a common cause of a P–R interval that is much prolonged without dropped beats. Other factors, however, are generally present, and the most important of these seems to be some lengthening of the P–R interval before digitalis therapy; even then, a concurrent infection is often the immediate cause of the dropped beats. In a series of cases of partial heart block nearly 40 per cent of those with dropped beats were being given digitalis, and 15 per cent of those with latent heart block.

The most essential prerequisite was some prolongation of the P–R interval before treatment. In those with partial heart block and dropped beats, and in those with latent heart block it averaged 0·21 sec. Only in three of twenty-three was the P–R interval below 0·18 sec.; in these three it was 0·16 sec., and in two of them there was acute infection also. When there were no dropped beats,

the P R interval was on the average increased to 0·26 sec. When there were dropped beats, there were most often two responses before the dropped beat, and the lengthening P–R intervals averaged 0·22 sec., 0·37 sec., dropped beat, etc. This, however, was the average of very varied figures, which averaged 0·22, 0·29, dropped beat, etc., in three, and 0·22, 0·41, dropped beat, etc., in six patients. No special difference could be found between the groups with these different responses. Apart from the presence of latent heart block the ætiology of the underlying heart disease did not seem of importance, though naturally congestive failure was present in the majority, as this was usually the indication for digitalis.

The presence of a concurrent infection seemed the next most important factor. When there were dropped beats, more than half had some active infection at the time. Sometimes this was a severe infection such as active rheumatic carditis, but often it seemed of a trivial nature, and it was only because digitalis could be taken at other times without producing dropped beats or because the rise of temperature ran so closely parallel to their onset that one could be sure of the connection. When there was latent heart block, an active infection was less often present, but was noted as the cause in a few cases. In others where there was no infection but a heart that was very seriously damaged (as shown by the patient's death within a relatively short time) this seemed an additional factor making the conduction time more sensitive to digitalis than usually.

Large amounts of digitalis were rarely the cause. Three of these cases were taking amounts that would generally be thought excessive (and curiously enough two of these had acute infections at the time and there seemed no reason why any digitalis should have been given); seven had amounts that were large, but quite reasonable; and twenty-two were taking amounts that were average or sometimes even small.

In general, there seemed to be no severe ill-effects, even when there were dropped beats; and the block passed off quickly (within two or three days), but, of course, this immunity is dependent on the heart block being recognized quickly.

There seems no reason why the chance finding of latent heart block should prevent adequate treatment with digitalis where this is indicated, though naturally the case should be watched even more carefully than usual.

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# BLOOD PRESSURE AND OLD AGE

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Twenty years ago, Thompson and Todd (1922) published an article entitled " Old age and blood pressure problems." They recorded the blood pressure in a hundred and two Chelsea pensioners over the age of seventy-five, noting that forty-six of them had systolic figures over 150 mm. and twenty-three over 170 mm. They also found that apparently similar men in normal health showed great differences in blood pressure without obvious cause.

Several other writers have published figures of blood pressure readings in old age and these show considerable variation in their estimate of the normal. For example, Thewlis (1941) quotes Saller as stating that the average figures for men and women between the ages of sixty and sixty-seven are 173/93 and 216/102 respectively, and between sixty-seven and eighty-nine 186/80 and 222/112 respectively. Willius (1931), on the other hand, taking seven hundred cases over the age of seventy-five, attending the Mayo clinic, has figures varying from 109/75 to 127/74 mm. as the averages of his (two-year) groups. Even so, 74 per cent of his patients had systolic figures over 140 mm. and 40 per cent diastolic figures over 90 mm. Robinson and Brucer (1939), who took normal cases for insurance purposes, Bowes (1917), Richter (1925), and Wildt (1912) all published figures for five-year age groups. These are shown in Table I below, together with those of the present series.

TABLE I

AGE AND MEAN BLOOD PRESSURE

| Age Groups | Robinson & Brucer | Richter | Wildt | Bowes | Howell |
|---|---|---|---|---|---|
| 60–64 | — | 138/74 | 137/76 | — | — |
| 65–69 | 139/75 | 150/71 | 143/78 | 151/82 | 149/80 |
| 70–74 | 137/77 | 150/73 | 148/80 | 160/86 | 157/83 |
| 75–79 | 154/80 | 155/68 | 153/82 | 166/86 | 166/87 |
| 80–84 | 147/87 | 157/69 | 148/78 | 175/84 | 158/84 |
| 85–89 | | 161/67 | 162/85 | 170/90 | 147/79 |
| over 90 | — | — | 129/59 | 142/81 | |
| Number of cases | 189 | 165 | ? | 150 | 120 |

All the above figures are lower than those of Saller but higher than those of Willius. They agree that a rise of pressure is customary after the age of sixty, reaching a peak somewhere between seventy-five and eighty-nine but falling

thereafter. More than this cannot be deduced without a careful analysis of the clinical material from which the figures were taken. The present study was an attempt to carry the work of Thompson and Todd a stage further by classifying the cases observed and trying to find some relationship between blood pressure, arteriosclerosis, mental state, and physical condition.

## Conditions and Standards

The investigations were carried out on Chelsea pensioners, all tough veterans of the old professional British army. Their ages ranged from 65 to 92 and they comprised all men admitted to the Infirmary of the Royal Hospital, Chelsea, during a period of six months. Each patient was classified by blood pressure figures, the state of the arteries, renal concentration power, mental state, and physical condition. The blood pressure was taken usually once a week, but more often if the condition of a patient was changing rapidly. The method used was that recommended by the American Heart Association and the Cardiac Society of Great Britain and Ireland.

Owing to the evacuation of the Infirmary into temporary quarters, due to enemy action, the normal sources of X-ray and pathological investigations were not available, so that clinical methods only were used.

*Variations in blood pressure.*—Weekly variations of pressure were frequent within narrow limits. For instance, one patient kept between 190/90 and 210/110 throughout the whole period under review. Another stayed between 120/70 and 140/80, and a third between 140/80 and 160/90. Systolic figures appeared to vary about 20–25 mm. and diastolic figures 10–20 mm., but while the patient remained in his usual state, they rarely changed more than this. No significant pressure difference was found between the right and left arm of any patient, including those who were hemiplegic.

Infections, especially those with diarrhœa or prolonged pyrexia, almost always caused a fall in blood pressure. This might be as much as 50 mm. (systolic) with pneumonia, 25 mm. with enteritis, and 20 mm. with bronchitis. Any form of cardiac failure also lowered the blood pressure, which might fall from 180/90 to 90/60 in a week. The later stages of cancer were also associated with a drop in pressure. One patient, observed over two and a half years, went from 200/90 to 115/65 before death, while another fell from 190/90 to 110/60 in a few weeks. This was observed in seven cases.

## The Blood Pressure

Three main groups were noted. The first was composed of those who remained above 160 mm. systolic throughout the period of observation. This was classed as raised blood pressure, and included 42 per cent of the total. The second consisted of those with systolic figures below 160 mm. but above 115 mm. The third was made up of those with systolic pressures under 115 mm. and included 5 per cent of the total. While these groups were arbitrary ones, nevertheless they corresponded with certain clinical classes.

*High blood pressure.*—Fifty of the patients had mean systolic pressures over 160 mm. during the period of investigation. Ten more, originally in this group,

dropped to lower levels with cancer, infections, or heart failure. (These cases either died or became invalids, instead of living a fairly normal life as they had done before.) Both ninety-two year olds were in this class.

TABLE III

AGE AND BLOOD PRESSURE

| Age | Total number | Systolic over 160 mm. | Diastolic over 100 mm. |
|---|---|---|---|
| Under 70 .. .. | 26 | 40% | 8% |
| 70–74 .. .. | 33 | 36% | 12% |
| 75–79 .. .. | 34 | 51% | 18% |
| Over 80 .. .. | 27 | 42% | 4% |

Thirty-three of the men were physically active and twelve more only slightly incapacitated. Mentally, eleven were of normal adult intelligence, twenty-five were of normal senile intellect, and fourteen had some mental impairment. Eighteen had very thickened palpable arteries, while only two had arteries that were relatively soft. Nineteen had a specific gravity of urine below 1015, but only two had albuminuria.

This group with raised blood pressure included many of the fittest men in the Infirmary. Eighteen of them had local disabilities, such as old injuries or impaired vision, which made them unsuitable for Chelsea with the possibility of air raids. The rest were suffering from such conditions as bronchitis, enlarged prostate, or mild senile dementia, while four were hemiplegic. The higher ranges of blood pressure were unusual, only eight patients having systolic figures over 200 mm. and only thirteen having diastolic pressures regularly over 100 mm.

*Low blood pressure.*—Six patients had systolic figures regularly below 115 mm. and five more had similar pressures for long periods. The only survivor of the first class was a case of colitis, the others all dying of cancer or cardiac failure. The temporary members of the group suffered from chronic bronchitis (2), pneumonia, septicæmia, or sciatica. In each case the systolic pressure rose to a level of 130–140 mm. on recovery.

## ARTERIOSCLEROSIS AND MENTAL CONDITION

*Arteriosclerosis.*—Each patient was classified according to the state of his arteries as slightly thickened (+), moderately thickened (++), or considerably thickened (+++). No case had soft vessels. Both radial, brachial, and temporal arteries were taken into consideration before grading. In fifty patients, the findings were compared with the state of the retinal arteries. Some local variations were often found, as in the case of a man of 84 whose right radial was considerably thickened, but whose other accessible arteries were just palpable, and the retinal vessels little thickened. Another, suffering from senile diabetes, had slightly thickened palpable arteries with a markedly arteriosclerotic fundus.

The oldest man with only slight thickening of the arteries was 84. Five of the group had normal adult intelligence, while seven had ordinary senile mentality.

Only one showed mental impairment—the diabetic referred to above. Eleven were active enough to walk in the grounds of the Infirmary and to go up and down stairs without difficulty. Only two had hypertension, while three sometimes had pressures below 115 mm. systolic. This seemed to suggest a connexion between soft arteries and good physical and mental condition.

TABLE III

ARTERIOSCLEROSIS AND BLOOD PRESSURE

| Arterial thickening | Toal number | Raised blood pressure | Lowered blood pressure | Mentally impaired |
|---|---|---|---|---|
| Slight .. .. | 13 | 16% | 24% | 8% |
| Moderate .. .. | 80 | 38% | 4% | 22% |
| Marked .. .. | 27 | 66% | 20% | 32% |

Twenty-seven had extremely thickened arteries. Each member of the group where the pressure was not raised was in a feeble physical condition, unfit for much exertion. Five were also mentally impaired. Eighteen normally had a raised pressure, six of them being over 80. Eight of them had intelligence below normal senile standards: none were above it.

Out of fifty cases whose ocular fundi were examined, some thirty showed arteries thickened and tortuous in the centre of the disc, narrowing peripherally. Many of these had some irregularity in the lumen of the vessels. Sixteen had arteries that were narrowed, but more or less straight, throughout their course, not showing any irregularities. Two seemed to be normal and two had vessels that were difficult to see clearly. The state of the vessels appeared to have no relation to the level of blood pressure or the general state of the arterial tree, thirteen of the first class and eleven of the second having a raised pressure.

Of those with marked arteriosclerosis, the patients with raised blood pressure were, on the whole, more active and alert than the rest. It seemed that, over the age of seventy-five, marked thickening of the arteries was not important prognostically, especially when the blood pressure was raised. Below seventy and with relatively low blood pressure, some mental and physical impairment was usual. The power of recovery from injuries and infections was certainly poorer than in those with raised blood pressure who were a few years older.

*Mental condition.*—The mental state of patients was assessed as normal adult, normal senile, or impaired. The first class is self-explanatory. The second included those who could behave rationally and carry on a sensible conversation, but whose perceptions were somewhat dull and whose mental processes were slower than those of younger persons. The third class was composed of those who were clearly abnormal or subnormal.

Fifteen patients had normal adult intelligence. All were physically active and three actually employed on light duties in the infirmary. The oldest member of this group was 75 years of age. Eleven had a raised blood pressure and ten showed moderate thickening of the arteries, while none had extreme thickening. None of these cases had urine with specific gravity below 1015.

TABLE IV

MENTAL CONDITION AND CIRCULATION

| Mental standard | Total number | With raised blood pressure | Arterial thickening + | ++ | +++ |
|---|---|---|---|---|---|
| Adult .. .. .. | 15 | 73% | 33% | 67% | — |
| Senile .. .. .. | 78 | 33% | 10% | 66% | 24% |
| Impaired .. .. | 27 | 50% | 4% | 66% | 30% |

The normal senile group included seventy-eight patients, of whom twenty-five had raised blood pressure. Nineteen had marked arteriosclerosis, but in seven the thickening was only slight. Sometimes a man in this class would go downhill and become mentally impaired, and this was usually associated with some intercurrent disease or change in blood pressure. For example, a patient whose general levels were between 120 and 150 mm. systolic became mentally worse on falling to 110 mm.; this was a mild case of the syndrome of progressive cerebral ischæmia (Howell, 1941).

Twenty-seven cases were mentally impaired. Fourteen of these had a raised blood pressure and two had pressures below 115 mm. systolic. Only one had slight thickening of his arteries and he had also an arteriosclerotic fundus oculi. Eighteen had moderate, and eight marked thickening of the arteries. Ten of the cases had a low specific gravity of urine, while only one had a figure above 1025. Six of these mentally impaired patients were hemiplegic. Thirteen were among the group whose fundus oculi was examined; nine had arteries thickened in the centre of the disc but narrowing peripherally, and the rest had vessels that were straight and narrow throughout their course.

## DISCUSSION

As we have seen above, a systolic pressure above 160 mm. is common in old age. In the five-year age groups examined, the proportion varied from a third to a half of all cases. Among the oldest patients, the fittest were those with a high pressure; and a figure below 125 mm. was only seen in three men over 80, two of whom were dying and the third of whom had exceptionally soft arteries for his age. The findings suggest that increased pressure is needed to offset the arteriosclerosis that becomes more pronounced with age (see Table V). Marked thickening of arteries with a relatively low blood pressure is associated with poor physical condition, while raised blood pressure and very thick arteries are compatible with relative fitness and activity. The connexion is not so clear in the case of mental impairment, but we can say that no man with very thickened arteries had adult intelligence and that no patient with impaired intellect had less than moderate arteriosclerosis.

Measurement of the specific gravity did not give much help in the assessment of a patient's condition, only 10 per cent of cases having a figure above 1025 for early morning urine. Robinson and Brucer (1939) suggest that old age depends on low weight, low blood pressure, and a slow pulse. Experience at the Royal Hospital, Chelsea, does not accord with this, however. Seven out of

TABLE V

AGE, ARTERIOSCLEROSIS, AND RAISED BLOOD PRESSURE

| Age | Total number | Arteries + | Arteries ++ | Arteries +++ | Patients with high blood pressure |
|---|---|---|---|---|---|
| Below 70 .. .. | 26 | 20% | 65% | 15% | 40% |
| 70–74 .. .. | 33 | 15% | 70% | 15% | 37% |
| 75–79 .. .. | 34 | 6% | 76% | 18% | 50% |
| Over 80 .. .. | 27 | 4% | 52% | 44% | 33% |

ten pensioners over ninety in recent years have been stout rather than thin: both ninety-two year olds in the present series had a systolic pressure habitually over 160 mm., while a pensioner, killed by enemy action at the age of a hundred, had a pressure of 150/80 during the last two years of his life. This man was also far from thin.

It seems that the raised blood pressure arising in old age is beneficent compared with that of younger persons. In the present series of cases, many of the younger men who had been admitted to the Royal Hospital as In-pensioners with high blood pressure, were in poor condition compared with their elders. As arteriosclerosis increases, a greater head of pressure is necessary to prevent ischæmia of vital organs or tissues, so that senile hypertension may well be compensatory in character. In the absence of raised pressure, vital ischæmia takes place, resulting in impaired function, either mental or physical. With the old man with raised blood pressure, cardiac defeat or a vascular accident eventually terminates life, but he is often spared the lingering existence, dependent on others for everything, that falls to the lot of some aged folk.

## SUMMARY

A rise of systolic blood pressure is common after the age of sixty.

In the present series of 120 Chelsea pensioners, 42 per cent had systolic figures regularly over 160 mm.

Cancer, cardiac failure, and infections often cause a fall in blood pressure.

Marked arteriosclerosis, in the absence of raised blood pressure, is usually associated with poor physical condition.

It is suggested that the raised blood pressure of old age is a form of compensation tending to prevent ischæmia of vital structures.

I should like to express my thanks to the nursing staff of the Royal Hospital, Chelsea, for their assistance in my investigations.

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# TRANSIENT INVERTED T WAVES AFTER PAROXYSMAL TACHYCARDIA

BY

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*From the Kilmarnock Infirmary*

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Graybiel and White (1934) have stressed that care must be taken in the diagnosis of serious heart disease when inverted $T_1$ or $T_2$ waves are the only abnormal finding, and that this inversion is not invariably associated with organic disease. They cited two cases of paroxysmal tachycardia in young robust adults, one followed by inverted $T_1$ and the other by inverted $T_2$ and $T_3$ waves; there was no sign of organic disease and there was a gradual return to a normal cardiogram. Campbell and Elliot (1939) record two cases of ventricular tachycardia that gave transient changes simulating coronary thrombosis; one of these gave a history of diphtheria and the other was of long standing and had been suffering from increasing dyspnœa during the intervals between attacks; both died suddenly at a later date. These cases show that the general condition must be given prime consideration in forming a prognosis.

It is generally recognized that the commonest causes of inversion of T waves are coronary thrombosis, myocardial disease due to toxæmia or hypertension, large doses of digitalis, the physiological effect of taking cold drinks, and the position of the heart. White (1941) has recently shown that change of posture may bring about an inversion of T waves especially in persons of the neuro-circulatory asthenic type.

## Present Case

A girl, aged 13 years, was admitted to Kilmarnock Infirmary with paroxysmal tachycardia that had already lasted 24 hours. The attack stopped spontaneously after a total duration of 36 hours; it was unaffected by any of the usual procedures to terminate it.

*Previous History.* An only child, nervous and of nervous parentage; whooping cough at 2 years but no sequelæ; no other ailments except chicken-pox, until her first attack of tachycardia three years previous to the present one, when she got a fright while on a swing; six attacks since then; her mother stated that these seemed to come on after an attack of flatulent indigestion but with no actual warning, the first thing the child felt being a " thumping " in her chest; the duration varied from 2 to 24 hours and each attack stopped suddenly, leaving the patient well but weak for some days.

*Examination.* On admission, pulse rate 214, respirations 26, and temperature normal. During the paroxysm there was an obvious impulse over the cardiac area; there was no enlargement of the heart; a systolic murmur most marked just to the left of the sternum; no signs of pulmonary congestion or of enlargement of the liver or spleen; but some flatulent gastric distention: a cardiogram confirmed the diagnosis of paroxysmal tachycardia, most likely of ventricular origin: radiological report, no abnormality either of heart or lungs. The next morning her pulse rate was 96 and the blood pressure was 112/82; she was comfortable but apprehensive; the cardiogram showed lowered S–T and inverted $T_2$ and $T_3$; on the next day it was the same; four days after the attack there was also some slight inversion of $T_1$; thereafter there was steady improve-

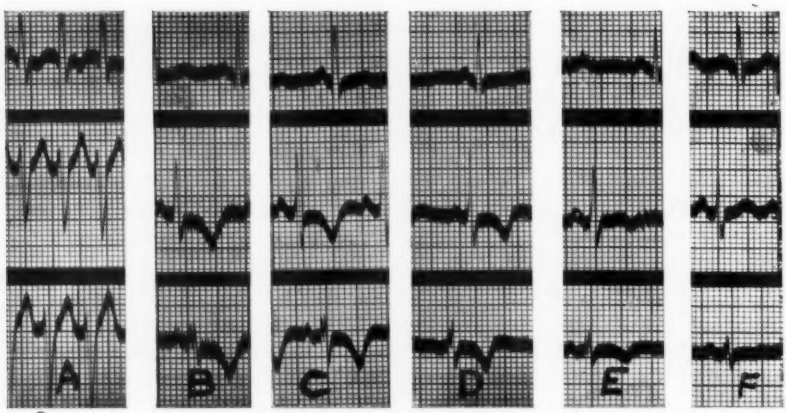


FIG. 1.—Attack on 31/10/39.
(A) During attack. (B) Following day. (C) 2 days after. (D) 4 days after. (E) 13 days after. (F) 30 days after.

ment till on 30/11/39 it was normal (Fig. 1). The patient was kept resting in bed for eight days and was dismissed at the end of two weeks.

On 4/6/40 re-examination showed a similar normal cardiogram; she had kept fairly well except for a morbidly introspective mentality for a girl of her years; she had been kept on a simple carminative mixture and small doses of phenobarbital.

On 28/8/40 she was seen at the start of another attack and a cardiogram taken within an hour was similar in form to the first but the T waves were higher; it should be remembered that the former had been taken 24 hours after the onset. This attack lasted three hours. The records in this case (Fig. 2) show that on 1/9/40 $T_2$ and $T_3$ were inverted but the S–T interval was unaffected; on 11/9/40 $T_2$ was isoelectric and $T_3$ only slightly inverted; on 25/9/40 there was a return to normal.

On 20/6/41 the cardiogram was normal; in the interval there had been no further attacks and the patient had much improved generally; she had been encouraged to lead a normal life; she mixed with other girls, and at the same

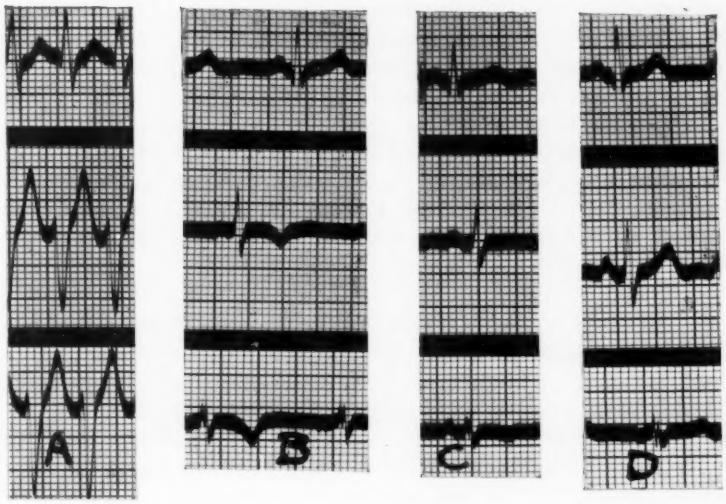


FIG. 2.—Attack on 28/8/40.
(A) During attack. (B) 4 days later. (C) 14 days later. (D) 28 days later.

time her mother had been prevailed upon not to shelter her only child so much; she was now working in an office.

At the present time (April, 1942) she is well and has had no return of the tachycardia since August, 1940; her pulse rate and blood pressure are normal, as is also the cardiac condition; her nervousness is decidedly less.

## DISCUSSION

When this patient first came under observation she was classed as neurocirculatory asthenia on account of her general state and the absence of organic disease and of any history of disease leading to cardiac complications; she was easily tired and excited, and had palpitation and dyspnœa with slight exertion and excitement, and slight tachycardia even when resting.

The degree of inversion of the T waves and the disturbance of the S–T interval appeared less after the second attack which was only 3 hours duration as against 36 hours in the first; this would seem to indicate that the duration and hence the degree of strain on the myocardium affect the amount of temporary change in the cardiogram; this is important when one comes to consider the cause of the inversion, and lends weight to the theory that it is due to overstrain of the myocardium.

The primary fact that emerges is that abnormalities simulating coronary thrombosis have arisen apart from detectable organic disease; if the paroxysmal condition had not been confirmed at the time of the attack, the subsequent records might have raised suspicion of serious heart disease. The second series confirmed the transient nature of the cardiographic abnormality. The case

has not been reported earlier in order that a sufficient time should elapse to rule out the development of cardiac complications. In view of the different endings in the cases quoted and the present one—fatal in those of Campbell and Elliot, apparent recovery in those of Graybiel and White and the present case—the clinical findings should be taken into consideration, when it is seen that a bad prognosis was probable in the fatal cases in view of the presence of organic lesion or toxic history, while in the other three cases there was no discoverable organic lesion. This is obviously another instance of a condition where negative T waves may not have the serious prognostic import usually attributed to them.

## Summary

A case of recurrent paroxysmal tachycardia in a girl with neuro-circulatory asthenia is described; at the time of first observation she was 13 years of age and is now 16; serial cardiograms during and after each attack showed transient inversion of the T waves and a lowering of the S–T interval in one series; there was a return to normal in each case in a month; the degree of the inversion is probably affected by the duration of the attack. The relative importance of the clinical over the cardiographic findings in giving a prognosis in such cases is stressed, and a possible cause of the inversion is put forward.

*Addendum.* Since this article was written Campbell (1942) has reported three cases in addition to those already referred to (Campbell and Elliot, 1939); these additional cases were of the clinically " benign " type, and the transient alteration in the T waves was considered not to " indicate any organic disease but to be a completely reversible process indicating some degree of exhaustion or strain of the heart muscle," a conclusion agreeing with that already formed regarding the present case.

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# PROGNOSIS IN PAROXYSMAL TACHYCARDIA AND PAROXYSMAL AURICULAR FIBRILLATION

BY

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In 1888, Bristowe wrote of " Recurrent palpitations of extreme rapidity in persons otherwise healthy "; he was, however, extremely guarded as to the correct prognosis in this condition, seeing that six out of nine of the cases he described died before their time. Similarly, two of the three cases recorded by Bouveret (1889) died of congestive failure during an actual paroxysm. Indeed the early reports are characterized by the number of fatal cases, so that it is not surprising to find Herringham (1897) writing " when one sees a robust and healthy person subject to these attacks, it is tempting to make light of them, but this ought never to be done. The condition seems always to tend to get worse and, in the end, to kill."

We do not in our generation take such a gloomy view of the attacks, but even now it is not uncommon to find patients, who are otherwise organically sound, living a life of a cardiac cripple following the pronouncement of a poor prognosis. We are, therefore, submitting our observations on a large number of patients with paroxysmal auricular tachycardia, especially on those who are otherwise perfectly healthy, in order that a more optimistic outlook shall be the rule to counteract the somewhat pessimistic attitude of earlier writers.

The basis of this paper has been chiefly the private case records of one of us (P.D.W.), these consisting of more than 600 cases of paroxysmal tachycardia. In addition, all those in the index files of the Massachusetts General Hospital between the years 1928–38 were reviewed. Numerous others from the cardiographic laboratory were also collected so that the total number of cases considered was more than 750. A separate paper has been submitted dealing with paroxysmal ventricular tachycardia.

## Immediate Prognosis of Paroxysmal Auricular Tachycardia

The likelihood of any particular attack ceasing, in spite of the most alarming symptoms, is practically certain. Cases are, however, encountered from time to time in which death takes place during an attack, and there were seven in this series in which death was due, directly or indirectly, to a paroxysm.

*Case* 16. A woman, aged 44, was admitted to the hospital on the seventh day of a paroxysm of auricular tachycardia with signs of congestive failure. Shortly after,

she had a femoral embolus and a little later, during a recurrence of her tachycardia, a cerebral embolus from which she died.

*Case* 18. A boy, aged 4, had had repeated attacks of tachycardia since the age of 2, on many occasions developing signs of congestive failure. Injection of the cervical ganglia was carried out with some success, but finally, after numerous and almost continuous paroxysms, he had a cerebral embolus and died.

*Case* 10. A man, aged 42, started with paroxysms of auricular tachycardia at the age of 16. At 26, he had joint pains and " heart trouble." The attacks of tachycardia became frequent, being associated with loss of consciousness, hæmoptysis, and pulmonary œdema on one or two occasions. Between the attacks he was able to work as a carpenter. He was finally admitted in an attack with acute pulmonary œdema from which he died. He had " tight " mitral stenosis.

*Case* 29. An obese woman, aged 48, developed paroxysmal auricular tachycardia the morning following bilateral salpingo-oophorectomy and hysterectomy. She became unconscious, with a blood pressure of 80/60, and died 8 hours after the onset. EC., paroxysmal auricular tachycardia, rate 180.

*Case* 30. A woman, aged 42, had had a difficult and prolonged convalescence following an operation for intestinal obstruction. Paroxysmal tachycardia set in 24 hours before death, and did not respond to any treatment. She died after the rapid development of failure (B.P. 60/30). Autopsy showed an apparently normal heart.

*Case* 31. A woman, aged 57, developed paroxysmal auricular tachycardia, at a rate of 220, with the picture of profound surgical shock. The temperature gradually rose, and she died in circulatory failure, 12 hours after onset. Her blood pressure was 110/80, her usual pressure being 200/100. Autopsy showed a normal heart.

*Case* 55. A boy, aged 2, started very frequent paroxysms of auricular tachycardia following pneumonia and whooping cough with the development of congestive failure. No treatment was of any avail and he died.

Very little comment is needed on these cases. Three of the deaths occurred in patients who were already sick from severe major operations. The tachycardia, with the production of profound shock, superimposed upon the trauma of the operation, merely proved to be the straw that broke the camel's back. Two of them, it will be seen, developed cerebral emboli. Just as there is always a possibility of cerebral emboli in auricular fibrillation so too the same possibility is always present in a paroxysm of tachycardia that continues for any length of time, especially when congestive failure develops. Case 10 illustrates the danger that is always present in those who have both mitral stenosis and paroxysmal tachycardia. Five others were encountered with mitral stenosis and severe pulmonary œdema from paroxysmal tachycardia, but without a fatal termination.

### The Remote Effect of the Paroxysms

Numerous individual cases in which attacks have occurred over several years have been reported (e.g. Palmer, 1937; Cowan, 1939). Hume (1930) collected a series of 130 cases in which the average duration was over 30 years, while Campbell and Elliott (1939) reported in their series of 100 cases, a group of 28 with apparently normal hearts whose paroxysms had been occurring for an average of 23 years. There were two deaths during the attacks in this group. There is no reported evidence to suggest that permanent organic heart disease may be produced by such repeated paroxysms.

In this survey, 80 cases with otherwise normal hearts were followed up. These, except for 4 hospital cases, were patients who had been seen in consultation and had had their paroxysms over a period of ten years at least. Of these 80, 57 were traced either by letter, through their own medical practitioner, or in person.

The average age of the group that has been traced was 58 years and the average length of time since the onset of the attacks was 25 years. The longest duration for any patient was 64 years. His attacks apparently caused no serious inconvenience and he classed them merely as a nuisance.

*Case* 32. A man aged 72, had had his first attack at the age of 7. Since then he had had 1 to 2 attacks of tachycardia for a year, varying from 6 to 24 hours in duration. When last seen, he had diabetes and mild coronary disease, and had 6 to 8 attacks a year which were well controlled with quinidine. He was still very active.

It was found that five of the patients had died, but there was no evidence to associate the paroxysms with the deaths of any of the first three patients.

*Case* 37. A woman, aged 52, had had paroxysmal tachycardia for approximately 25 years. She died following an operation for gallstones, but the exact details were unknown.

*Case* 38. A man, aged 69, had had 6 to 8 attacks a year for 32 years. He died of pneumonia despite a week in an oxygen tent.

*Case* 39. A woman, aged 75, had had paroxysmal auricular tachycardia for 15 years. The exact cause of death was not known.

Case 40. A man, aged 55, had had paroxysmal auricular tachycardia for 40 years. Over the last 4 years of his life, he suffered with symptoms of hypertensive coronary heart disease. The paroxysms became very much more frequent and congestive failure was precipitated. He died at home, but the details could not be obtained.

Case 26. A woman, aged 50, had had paroxysmal auricular tachycardia for 18 years. It had been extremely troublesome the whole period, at times causing slight cardiac enlargement. She died following an operation for carcinoma of the breast. Autopsy showed a recent auricular infarct.

Unfortunately no adequate histological studies had been carried out on this case and the heart was not available for re-study. The patient herself had been otherwise healthy and able to carry out her duties as a nurse. One can only speculate as to what modifications there may have been in the blood supply of the S-A and A-V nodes. The abnormalities of rhythm during the paroxysms in this case have been reported by Sprague and White (1925).

Thus, concerning the prognosis in patients with paroxysms occurring over many years, there seems to be no conclusive evidence in this small series to justify any adverse statement and, indeed, medico-actuarial figures suggest that such patients have a slightly better expectation of life than normal.

According to Campbell and Elliot, the prognosis of paroxysmal auricular tachycardia " as regards life is excellent, unless appearing relatively late in life it is the first indication of disease of the coronary arteries or unless before the paroxysms have started there is already serious heart disease. In general, the prognosis depends on the condition of the heart muscle and should be decided

without reference to the paroxysms." It is not, however, our experience that the prognosis is poor, even coming on late in life, and the following two cases are examples of the relative insignificance of attacks in the majority of such patients.

*Case* 34. A woman, aged 78, began to have rather frequent attacks at the age of 56. These continued until about three years ago when they practically disappeared. She now writes of her excellent health—no doctors, no medicine, and the ability to undertake five-hour train journeys by herself.

*Case* 35. A woman, aged 80, started to have attacks when 56, with occasional paroxysms lasting three to six hours. She now has slight hypertension, but is quite active.

Moreover, a survey of 398 consecutive cases of paroxysmal tachycardia seen in consultation showed that there were 69 with coronary heart disease and/or hypertension, with the onset of the arrhythmia after the age of 40. Included among these there were 16 patients with coronary thrombosis. Out of 128 patients with otherwise normal hearts in this same series, 57 started their attacks after the age of 40. Thus, excluding the cases of coronary thrombosis, there were approximately equal numbers with and without organic heart disease starting after the age of 40. The whole of this group has not been followed up, but a large number of the patients had been re-examined from time to time; most of the remainder were first seen four to five years after the onset of the tachycardia. These patients, therefore, form a group from which it is possible to draw some conclusions as to the prognostic significance of tachycardia arising in the second half of life.

Two patients of this older group with apparently normal hearts died unexpectedly. The first died following an operation for gallstones at the age of 52; and the second, a man, also aged 52, died while swimming, one year after he had been diagnosed as having an irritable normal heart. In neither of these were the exact details as to the mode of death available. Even taking into consideration these two deaths, the impression was left that only in a very few of the cases with apparently normal hearts was paroxysmal auricular tachycardia the first symptom of serious organic heart disease.

In the group that already had coronary disease, the attacks of tachycardia did not appear to make any appreciable difference to the ultimate prognosis.

Master, Dack, and Jaffe (1937) studied the arrhythmias that were found in 300 cases of acute coronary thrombosis and found 9 (3 per cent) with paroxysmal auricular tachycardia. Only 2 died (a result of the thrombosis), and it was considered that patients with paroxysmal auricular tachycardia usually survived.

Frequent paroxysms might justify a more serious prognosis in a damaged heart than the organic disease itself warrants. It would be logical to suppose that a severe paroxysm of auricular tachycardia with its attendant low blood pressure might easily precipitate a coronary thrombosis. Indeed, the descriptions in some of the earlier reports (e.g. Bristowe, Bouveret) suggest this possibility. In none of the cases in our series was there any evidence to show that such an event had taken place. However, it might be exceedingly difficult to determine which occurred first, the tachycardia or the thrombosis.

To sum up, paroxysmal auricular tachycardia arising in later life, may warrant a more serious prognosis in individual and comparatively uncommon cases, but as a general rule, the attacks have little or no influence on the ultimate outcome.

*Prognosis as to the frequency of the attacks.*—It is difficult to give any forecast as to the possibility of gaining complete freedom from the attacks. Some patients, such as Case 41, had attacks every two to three weeks and became very disheartened because the attacks showed no improvement and, as the years went by, became very much more frequent: while others, such as Case 42, suddenly had complete remission. This patient had increasingly frequent attacks for five years until the tachycardia became almost constant and signs of congestive failure were present: then following treatment with quinidine, she has been entirely free from attacks for fourteen years.

The patient with the most frequent attacks was Case 43, who stated that he had as many as 10 to 12 attacks in a day and that the attacks had been present for the past 25 years.

The prognosis of any given case is inextricably bound up with treatment. Some patients seem to undergo a spontaneous cure, others react to specific treatment, and a number continue unchecked by any measure whatsoever. It seems impossible to foretell in what way a given case will react. As a rough guide, the patients who are extremely neurotic and have developed a marked degree of neurocirculatory asthenia, have a poor chance of getting rid of the attacks.

*Duration of the attacks.*—Attacks of long duration have been recorded by many writers (Bouveret; Bristowe; Hamilton and Hurvitz, 1932; Gallavardin; Veil and Froment, 1930; Marvin, 1923). It has also been emphasized frequently that such attacks of long duration are uncommon. One infant, aged 18 months, was seen in whom paroxysmal auricular tachycardia had been present for over twelve months; she was otherwise a perfectly normal and well-developed child.

Lewis (1936) states that the attacks in each individual patient are usually of the same duration. We have found, however, that the duration of the attacks is extremely variable. It is, therefore, impossible to give a forecast of the length of any future paroxysm, although one can usually safely say that paroxysms of more than 24 hours in duration are uncommon.

## Prognosis in Paroxysmal Auricular Fibrillation

Parkinson and Campbell (1931), as a result of their review of 200 cases of paroxysmal auricular fibrillation, considered that the prognosis in the immediate attack was excellent as it would seldom last more than two days, and that the ultimate prognosis depended mainly on the condition of the heart muscle independently of the paroxysms; though these increased the risk of fibrillation becoming established, which happened in almost one quarter of all the cases and in a higher proportion of those with rheumatic heart disease or with very frequent paroxysms. They made no actual statement, however, as to the

prognostic meaning of attacks in a damaged heart, whether such attacks would mean a more serious prognosis than a similar heart without such attacks.

White and Bland (1931) in a report on the prognosis of angina pectoris and of coronary thrombosis, found that the incidence of paroxysmal auricular fibrillation was low in both conditions. In the 500 cases of angina, there were only 11 examples and 4 others in which the attack occurred as a terminal event. In the 200 cases of coronary thrombosis, attacks of fibrillation occurred at the time of the actual thrombosis in 13: of these, 4 died after one month and within one year, and 5 others were still alive after four years. From their experience, fibrillation did not seem to increase the gravity of the prognosis.

Master, Dack, and Jaffe (1937) in their 300 cases of acute coronary thrombosis, found 22 with fibrillation. In half these there had been a previous paroxysm. The average age of the group was 60 and the males predominated in the ratio of 2 to 1. In 18 there was a history of previous hypertension. 10 cases died, 5 during the period of the arrhythmia. Comparing the mortality of the cases with and without arrhythmia in acute thrombosis, they found a slightly increased mortality in the cases with fibrillation. They concluded that the relatively low incidence of abnormal rhythms was due to the copious blood supply to the A-V node protecting it from serious damage.

We found the incidence of auricular fibrillation in a consecutive series of 1400 cases of angina pectoris and coronary thrombosis to be small, only 4 per cent. Many of these, who had coronary heart disease when first seen, were followed for several years until they finally died in a ripe old age. In none of them could it be said that auricular fibrillation had accelerated death. For example, one patient, a physician, had had attacks every three to six months for the past 28 years and was still active at the age of 83, though he had also intraventricular block. Another example is Case 47, who was still active though restricted at the age of 73 after attacks over the previous 14 years. One woman, aged 76, who had had attacks for the past 5 years, successfully underwent an operation for carcinoma of the breast.

In 1936, Orgain, Wolff, and White reported on the prognosis of paroxysmal auricular fibrillation occurring in otherwise apparently normal hearts. They were able to follow up 40 cases out of a group of 46. They concluded that the prognosis for life and for the maintenance of adequate cardiac function was, with rare exceptions, excellent. These cases with the addition of numerous private and of 4 hospital cases give a series of 88 cases. The diagnosis of paroxysmal auricular fibrillation and a heart otherwise normal had been made in every one of the 88 cases. The abnormal rhythm had been diagnosed by a physician in 26 cases, by a characteristic history in 10 cases, by physicians on themselves in 3 cases, and in the remaining 49 cases by electrocardiogram. Of these, 78 were traced either in person, by letter, or through their local practitioner.

Of the 78 cases that were traced, 22 were found to have died. The ages of onset and at death of these cases, and comments as to the mode of death, have been listed in the appendix. It will be seen that 6 died with auricular fibrillation complicating toxæmia, operation, or infection; 7 died of cardiac causes, chiefly

coronary thrombosis; 6 died unexpectedly; and 3 died of causes probably not connected with the heart, most likely of cancer. In none of the 4 cases of coronary thrombosis was there an autopsy. In the 6 who died unexpectedly, one must assume that there was in each case a cardiac cause, most probably coronary thrombosis. There were only 2 who were under the age of 40 at the time of death, and they were both given to alcoholic excess.

The 56 living patients may be divided into groups for the better estimation of the prognosis: 13 of the survivors were physicians and will be considered separately.

(*a*) *Age of onset between* 40 *and* 60, *and the time since onset more than two years.*—This group contained 23 cases, the average age of onset being 49 years and the average duration since the onset being 12 years. The number of other cases in this age group who had died was 16, of whom 4 died following operation or infection. Excluding these 4, the average duration from onset to death of the remaining 12 was 13 years, and the average age of onset approximately the same as for the whole group. Of the 23 alive, 6 developed signs of coronary insufficiency, either clinical or electrocardiographic. These developed 16, 15, 10, 8, 6, and 4 years after the onset of the paroxysms.

In this group, paroxysmal fibrillation may be the first signs of a serious heart disease, but there does not seem to be any evidence to suggest that the prognosis is any other than that of the underlying heart disease when it becomes evident.

(*b*) *Age of onset below* 40, *and duration more than two years.*—This group contained 16 patients that were living, with an average age at onset of 26 years and duration of 19 years. Excluding one who died of infection and alcoholism, 3 others died after 16, 13, and 8 years, respectively. Only one of those alive appears to have developed cardiac insufficiency, 32 years after the onset, at the age of 70.

The prognosis in this group appears to be excellent.

(*c*) *Physicians.*—This group contained 14 physicians. Only one of these has died. He developed hypertension and permanent auricular fibrillation, eventually dying of carcinoma of the œsophagus 24 years after the onset. The average age of onset was 42 years, and the average duration, in 12 of the doctors who had had attacks for more than two years, was 24 years. This high incidence in doctors was noted by Parkinson and Campbell. It suggests that the condition is more common than is usually supposed and that the prognosis is good.

(*d*) *Miscellaneous group.*—One man whose attacks came on at the age of 75 was still alive at the age of 86 although his exact condition was not known. In this group there were 3 patients with the abnormal complexes of the type described by Wolff, Parkinson, and White. They were all well after 1, 8, and 18 years, respectively. There were 9 patients who had only one attack. These were still healthy.

To sum up, the occurrence of paroxysmal auricular fibrillation should be regarded in general as having no serious prognostic significance, but when occurring in patients over the age of 40, it may be the first signs of organic

heart disease, in which case the prognosis given will depend upon the appearance and severity of the coronary heart symptoms.

*Frequency and duration of paroxysms.*—As in paroxysmal auricular tachycardia, it is impossible to give any forecast as to either the frequency, the duration, or the prospects of gaining complete freedom from the attacks. In those getting paroxysms with increasing frequency, permanent fibrillation eventually sets in. Occasionally, however, the paroxysms will be frequent for four or five years, causing very great inconvenience and discomfort, and no measure will give any relief until the onset of permanent fibrillation.

In rheumatic heart disease, it is usually thought that paroxysmal fibrillation heralds the approach of permanent fibrillation in the near future. In one patient, however, attacks were frequent for 13 years before this happened. Another had had attacks for 13 years; she was still working, having both mitral and aortic rheumatic disease. Yet another, with a similar heart lesion, had had paroxysmal auricular tachycardia for 10 years, and then for the last 3 years paroxysmal auricular tachycardia and auricular fibrillation. She recognized the difference in rhythm by the difference in the " position of the palpitation " in her chest.

A patient with a probable inter-auricular septal defect had her attacks for the greatest period of time without the occurrence of permanent fibrillation. They started when she was 13, and when last seen she was 58; her activities then were severely curtailed by angina pectoris.

The actual attacks themselves are short. In over half of the total series of 88 cases, they lasted less than 24 hours, and of these the most usual duration was less than 12 hours. Parkinson and Campbell consider that attacks lasting over seven days tend to be permanent, unless treated with quinidine. This is probably true, but with the introduction of quinidine, the outlook is changed. Case 51 had frequent paroxysms of fibrillation, one of which lasted for over three months before he was treated with quinidine. He was well nine years later, with normal rhythm, and was exceedingly active professionally. There were several cases of a similar nature in this survey. With an adequate maintenance dose of quinidine, the prognosis for the continuation of normal rhythm is good.

The prognosis of paroxysmal auricular flutter is essentially that of paroxysmal auricular fibrillation. Orgain, Wolff, and White (1936) recorded 7 cases in patients otherwise normal. It was possible to trace 6 of these who were still alive and well—29, 18, 7, 17, 32, and 9 years, respectively, after the onset. These findings confirm the good prognosis given in the original paper.

## Summary and Conclusions

When the cardiovascular system is normal, attacks of paroxysmal tachycardia are uncomfortable, but relatively unimportant incidents.

The probability of any attack ceasing is extremely good, but the occurrence of an attack in a patient seriously ill must be regarded with some apprehension, and the occurrence of heart failure as a result of the attack may lead to the formation of intracardiac thrombi and subsequent emboli.

The occurrence of paroxysmal auricular tachycardia in apparently healthy persons seems to have no effect upon the prospect of their longevity. When occurring late in life, the prognosis, in general, is that of the underlying heart disease, if present.

Paroxysmal auricular fibrillation is probably common. When occurring in patients below the age of 40, it may be considered as having no prognostic significance, while in later life the occurrence of fibrillation may, in some cases, be the first sign of serious heart disease.

It is impossible to forecast with any certainty either the frequency or the duration of the attacks of paroxysmal auricular tachycardia and fibrillation.

The basis of this paper formed part of a thesis submitted by one of us (W.T.C.) and accepted for the degree of M.D. of the University of Cambridge. The work was carried out while W.T.C. was holding a Research Fellowship in Medicine, Harvard University, and the Walter Myers Travelling Studentship, Birmingham University, 1938–1939.

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## APPENDIX

Deaths that have Occurred in 22 out of 78 Cases with Paroxysmal Auricular Fibrillation and Hearts otherwise Normal.

| Sex, and Age at Death | Age at Onset, | | Comments as to Mode of Death, etc. |
|---|---|---|---|
| M. | 53 | 76 | Apparently enjoyed excellent health until two hours before death, which was assigned to coronary thrombosis. Had taken daily quinidine which controlled paroxysms. |
| M. | 59 | 71 | Took quinidine, 6 grains daily, and had occasional paroxysms. Died of coronary thrombosis following golf. No previous symptoms suggestive of coronary insufficiency. |
| M. | 54 | 74 | Angina pectoris one year before death, and much more frequent attacks of paroxysmal fibrillation. Died suddenly, but no details available. |
| F. | 57 | 66 | Asthma for thirty years, and died during an attack. Post-mortem: recent thrombosis in the left ventricle. |
| M. | 44 | 62 | Last three years of life, under treatment for hypertensive coronary heart disease; rhythm normal all this time. Autopsy: old coronary thrombosis. |

| Sex, and | Age at Onset, | Age at Death | Comments as to Mode of Death, etc. |
|---|---|---|---|
| F. | 49 | 70 | Died of carcinoma of stomach, attacks of fibrillation having continued without other cardiac symptoms until death. |
| M. | 35 | 59 | Hypertension and permanent auricular fibrillation for last five years of life. Died from carcinoma of œsophagus. |
| F. | 45 | 65 | Died after an acute intestinal infection and signs of mental upset. Her doctor stated: no evidence that heart played any part in her death. |
| F. | 48 | 51 | Quite well for three years prior to her death. On returning to work following pneumonia, she dropped dead. |
| M. | 52 | 68 | Free from attacks for the last seven years of his life. Died unexpectedly, but details not known. |
| F. | 64 | 68 | Died following a convulsive seizure and a number of attacks of congestive failure. Fibrillation not present at time of death. |
| F. | 29 | 42 | Died with acute pulmonary œdema following an epileptic attack. No other details available. |
| F. | 44 | 53 | Died suddenly three years after being passed as having an irritable normal heart. Permanent fibrillation was thought to have set in. |
| F. | 48 | 51 | Follow-up study revealed she was dead, but details unknown. |
| M. | 27 | 35 | Died in congestive failure, with permanent auricular fibrillation. Alcoholic excesses played a major role. |
| F. | 52 | 56 | Asthma for twenty years. Died during a paroxysm of auricular fibrillation following a severe septic throat. |
| F. | 51 | 51 | Paroxysm of auricular fibrillation during erysipelas. Normal rhythm restored with quinidine, but died three months later, details unknown. |
| M. | 53 | 53 | Right-sided empyema for one year. Developed auricular fibrillation when this was reopened and died ten days later. Autopsy showed normal heart. |
| F. | 58 | 58 | Developed paroxysmal auricular fibrillation following an operation for rectal malignancy. Autopsy showed normal heart and lungs. |
| F. | 66 | 66 | Developed a severe sore throat a fortnight before death. Collapsed with onset of auricular fibrillation and died ten days later. Autopsy showed normal heart. |
| F. | 57 | 57 | Collapsed following an operation. Had auricular fibrillation for two days and died two days later. Autopsy showed normal heart. |
| M. | 35 | 35 | Developed auricular fibrillation following exposure and alcoholic excess. Died of tetanus. |

# FAINTING AND FITS IN CARDIAC INFARCTION

BY

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Thoracic pain, the cardinal symptom, may be absent in acute cardiac infarction, the best known equivalents being dyspnœa or other signs of congestive failure, and more rarely abdominal and cerebral syndromes. With pain of great intensity, syncopal symptoms may occur also as a result of cerebral vascular complications, and sudden death, which is not infrequent in this condition, can be regarded as the extreme form of syncopal attack. But occasionally disturbances of consciousness are a most prominent feature of the attack apart from all these conditions, with pain absent or displaced into the background. This is especially so at the onset of the attack. Many papers on cardiac infarction without much or any pain have referred mainly to patients with congestive failure, and disturbances of consciousness have not often been mentioned. (Bean & Read, 1942; Boyd & Werblow, 1937; East & Bain, 1928; Gorham & Martin, 1938; Gross & Sternberg, 1939; Hay, 1933; and Kennedy, 1937.) In the present series, seen among some 200 cases of acute cardiac infarction, syncopal symptoms were prominent in 15, in 10 at the onset and in 5 in the course of the illness.

## Syncopal Attacks at the Onset

Of 10 patients in whom cardiac infarction was ushered in by a syncopal attack (Table I) 5 were men and 5 women. The average age was advanced, five being 70 or more. Consciousness was partly or completely lost in all, but no convulsion was observed. The patient's appearance was that of severe shock or peripheral circulatory failure, the radial pulse being usually imperceptible, the skin pale and moist, and vomiting almost invariable. Of eight patients in whom the heart rate was known during the syncope, it was slow (50 or less) in five, in two there was sinus tachycardia, and in one with ventricular tachycardia the rate was 160. No cardiogram was obtained during these initial symptoms, but in one instance (Case 7) in which sinus bradycardia was recorded, pallor and a complaint of faintness while the tracing was being taken coincided with a drop in rate to about 24 a minute. In most of the other cases cardiograms taken between the second day and sixth week of the illness showed abnormal rhythms. In one and probably in a second there was A-V nodal rhythm, in two auricular flutter, in one auricular fibrillation, and in one ventricular tachycardia. The site of infarction could be located in nine; six in the posterior wall, three in the anterior wall, and the remaining

one probably also of the anterior type. Pain was absent or slight in five, moderate in four, and severe in one; in two it was confined to the abdomen. Six patients died between the first day and the fourth month, three from congestive failure, one after a hemiplegia, one suddenly, and one with symptoms of shock. The four survivors, who have lived an average of about three years since the attack, were with one exception the youngest of the group. None of these patients had previously suffered from any serious cardiac symptoms. In two there had been previous symptoms of cerebral origin and in five a high blood pressure reading was obtained at some time.

TABLE I

SYNCOPE AT ONSET IN CARDIAC INFARCTION

| Case No., Sex, and Age | Attack | Rhythm Recorded | Site of Infarction (*above*) and Degree of Pain (*below*) | Result |
|---|---|---|---|---|
| 1. M./72 | Unconscious; no pulse; heart 48. | Nodal rhythm (8 days later). | Anterior. None. | Failure. Died 8th day. |
| 2. M./62 | Fainting; no pulse; heart 35. | ? Nodal rhythm; short runs V.P.T. (six weeks after onset). | Posterior. Moderate. | Failure. Died 7th week. |
| 3. F./79 | Fainting; ? heart rate. | Auricular flutter (2nd day). | Posterior. Severe (abdomen). | Hemiplegia. Died 15th day. |
| 4. M./44 | Fainting; ? heart rate. | Auricular flutter (20th day). | Posterior. None. | Well 2 years later. |
| 5. F./72 | Fainting ; no pulse; heart 44. | Auricular fibrillation (8th hour). | Anterior. Moderate. | Died suddenly 1st day. |
| 6. F./55 | Fainting; pulse 160. | Ventricular tachycardia (4th day). | ? Anterior. Slight (abdomen). | Failure. Died 4th month. |
| 7. M./45 | Fainting. | N.R. rate 33. | Posterior. Slight. | Well 1 year later. |
| 8. F./70 | Fainting; pulse 50. | N.R.; rate 75 (10th day). | Posterior. None. | Well 3 years later. |
| 9. M./53 | Fainting. | N.R., S.A.; rate 70 (2nd day). | Posterior. Moderate. | Well 5 years later. |
| 10. F./82 | Fainting. | N.R.; rate 120 (2nd day). | Anterior. Moderate. | Died 3rd day. |

N.R.—Normal rhythm. S.A.—Sinus arhythmia.
V.P.T.—Ventricular paroxysmal tachycardia.

*Case* 1.* An active man of 72, previously well except for moderate hypertension and some transient cerebral symptoms one year before, was found unconscious just after he had gone to bed. When his doctor arrived, radial pulse imperceptible, some vomiting, heart rate about 48, and sounds only just audible. Next day the rate rose to 55 and remained there until death. Congestive failure developed within a day or

* All patients were seen in private practice and post-mortem examination was not possible in any.

two. The rhythm (Fig. 1) has been classed as nodal but resembles those recorded from subjects free of heart disease with physiological short P–R bundle branch block curves. Any of the interpretations that have been suggested for such curves would apply equally to this one. There are changes in the RS–T segments suggesting anterior infarction, though in lead IVR they are atypical.

*Case* 2. A man, aged 62, fell down in a faint when walking home. When seen by his doctor, he was sweating, bluish-gray in colour, some epigastric pain, radial pulse imperceptible, heart rate about 35. After vomiting, condition improved and next day perfectly well; heart rate still 35. After ten days allowed up but ten days

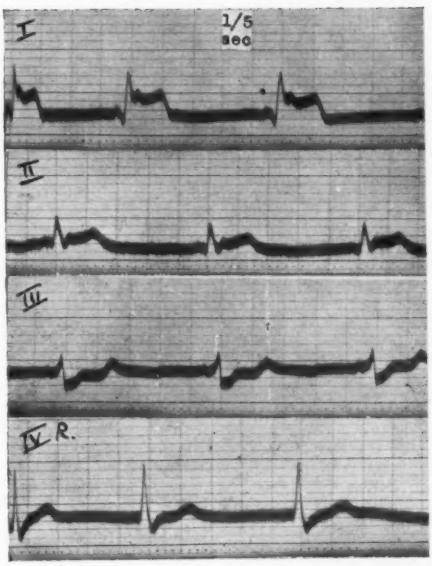



FIG. 1.—Case 1. Taken on the eighth day, a few hours before death. Regular rhythm, rate 60; short P–R interval, wide QRS complex.

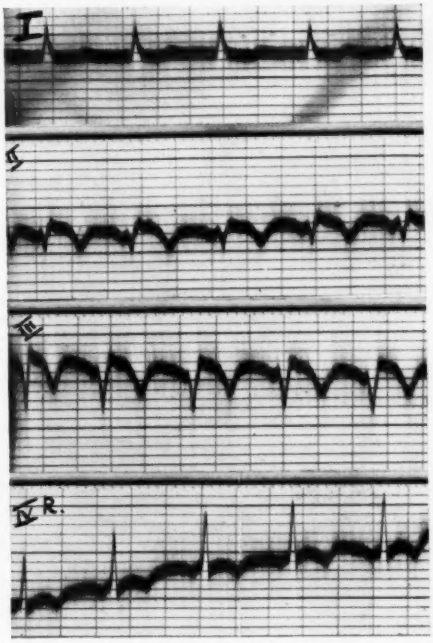



FIG. 2.—Case 4. Taken on the twentieth day. Auricular flutter (A. about 230 with 2 : 1 block); RS–T and T changes suggesting posterior infarction.

later some abdominal discomfort, dyspnœa, œdema in the feet and back, and rales at the lung bases. In the sixth week, the B.P. was 180/90, and there was severe orthopnœa and systemic venous congestion. Cardiogram: a basal ventricular rhythm, probably nodal, rate of 60 to 70, P wave absent; ventricular extrasystoles, some interpolated, sometimes multiple; left axis deviation, QRS low voltage; RS–T depressed in lead I, elevated in lead III; posterior infarction. Two days later the patient vomited and died suddenly.

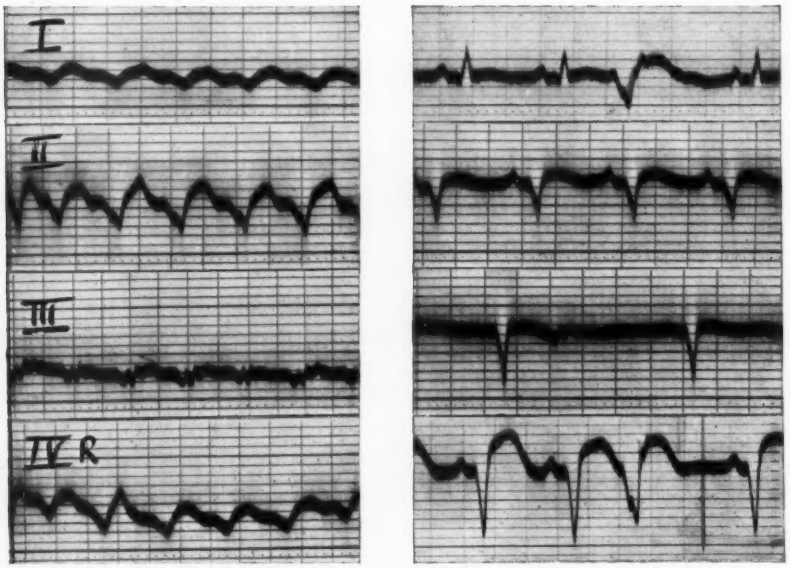
*Case* 3. A woman of 79, previously quite well, fainted in a lavatory. When seen by her doctor, severe upper abdominal pain, which next day was felt only in the left iliac fossa; there was dyspnœa, cold skin, sweating, and cyanosis; many fine rales at the left lung base; B.P. 120/100. Cardiogram: auricular flutter, A. 240 with variable 2 : 1, 3 : 1 block, right axis deviation; elevation of RS–T segment in all leads, $T_2$ and $T_3$ inverted, $Q_3$ present. Posterior infarction. Good progress until

the twelfth day when a sudden left hemiplegia was followed by her death three days later.

*Case* 4. A healthy man of 44 felt short of breath while playing golf; later became faint, vomited, and had a cold sweat. During next few days, slight icteric tint of the skin, systolic B.P. under 100, and some dyspnœa. Fig. 2 suggested posterior infarction. He was given digitalis, and 13 days later cardiogram showed normal rhythm, rate 85, P–R interval 0·35 sec., frequent ventricular extrasystoles some interpolated, RS–T deviation and T inversion less. Good progress, and well two years later.

*Case* 5. A woman of 72, previously well, fainted and then vomited. When seen by her doctor soon after, radial pulse imperceptible, but later palpable, rate 44; complained of pain behind mid-sternum, in jaw, and in left arm. Cardiogram taken nine hours after onset showed auricular fibrillation, ventricular rate about 165, QRS complex wide, R absent in lead IVR; RS–T deviation upward in leads I and IVR, downward in leads II and III. Anterior infarction. A few minutes after this was taken she died suddenly.

*Case* 6. A woman of 55 with hypertension for some years, hemiplegia two years before, and transient blindness one year before. In bed with epigastric pain for two or three weeks. Sudden faint attack with ashen colour, heart rate of 160, and orthopnœa. Four days later, Fig. 3 showed ventricular tachycardia, and two days




A B

FIG. 3.—Case 6. (A) Taken on the fourth day. Ventricular tachycardia, rate 155. (B) Two days later after 15 grains of quinidine. Normal rhythm, rate 95; R wave absent in leads II, III, and IVR; upward deviation of RS–T in all leads; occasional ventricular extrasystoles.

later after quinidine, normal rhythm. Periodic dyspnœa during the following weeks with a right pleural effusion. Then œdema of the legs which cleared up, but after increasing weakness the patient died in the fourth month.

*Case* 7. A man of 45 had some sternal discomfort after a day's shooting; he slept well and felt well next morning but fainted after a bath. When seen by his doctor, colour gray and some upper sternal discomfort; pulse rate was 40. Six hours

later comfortable, apex beat much out and heaving; B.P. 140/80. Fig. 4 showed sinus rhythm, at rate of 33 a minute. During the tracing heart rate varied and at the slower rate (under 24), pallor and faintness. Cardiogram nine weeks later, similar except for rate of 75, no deviation of RS–T segments, $T_2$ just negative, $T_3$ inverted. Well 15 months after the attack.

*Case* 8. A woman of 70 fainted without obvious cause; no pain but some sensation of wanting to defæcate; when seen, pulse rate 50 regular. Known to have had B.P. of 220/100 four years before. On the tenth day, comfortable, with no signs

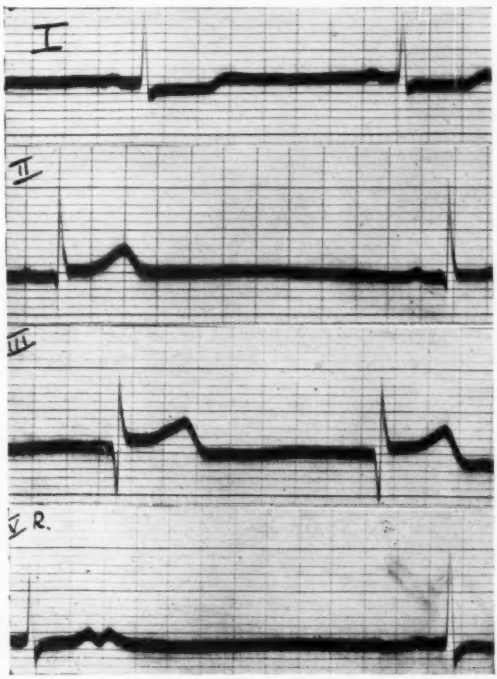



FIG. 4.—Case 7. Sinus rhythm, rate 33; P–R interval 0·20 sec.; P wave notched; small $Q_2$, large $Q_3$, QRS complex rather wide; RS–T depression in leads I and IVR, elevation in II and III; suggesting posterior infarction.

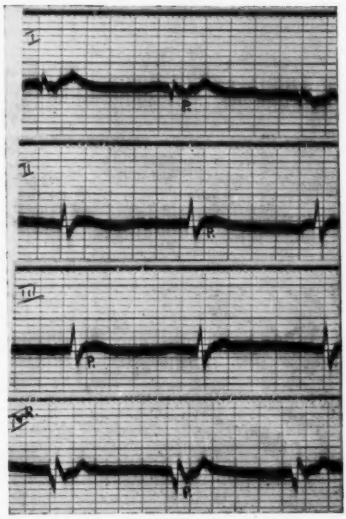



FIG. 5.—Case 11. Taken ten hours after the onset. Rhythm almost regular, rate 55; QRS, 0·1 sec.; negative P following QRS; RS–T slightly depressed in leads I and IVR, slightly elevated in leads II and III. Nodal rhythm; intraventricular block; posterior infarction.

of failure; B.P. 230/110. EC. showed normal rhythm, rate 80, QRS complex 0·09 sec., slight depression of RS–T in leads I, II, and IVR, $T_2$ diphasic, $T_3$ inverted; small $Q_3$; occasional ventricular extrasystoles. Three years later fairly well but auricular fibrillation; $Q_3$ no longer present and $T_2$ and $T_3$ upright.

*Case* 9. A man of 53, a chronic alcoholic, had fainted and then complained of upper sternal pain. When seen immediately by his doctor, sweating and colour ashen; pulse 68, regular. Next day felt well; B.P. 110/80, liver enlarged, no signs of failure. Cardiogram: normal rhythm, rate 70, QRS complex low voltage, $T_2$ just negative, $T_3$ inverted. Posterior infarction. Good recovery and no cardiac symptoms five years later.

*Case* 10. A woman of 82 fainted and had pain between shoulders with radiation into both arms. In bed next day, comfortable except for aching in back. No

dyspnœa; apex beat just out, loud systolic murmur at apex; B.P. 80/60. A cardiogram showed normal rhythm, rate 120 a minute; a very small R in lead IVR; slight depression of RS–T in leads II and III, and elevation in IVR; T wave in leads I and IVR just negative; suggests anterior infarction. Death occurred on the third day.

### Syncopal Attacks in the Course of Cardiac Infarction

Observations were made on five patients with disturbances of consciousness in the course of cardiac infarction that had begun in a typical way with anginal pain (Table II). There were four men and one woman, and the average age was 69. In two there was loss of consciousness without any fit; the other three had Stokes-Adams attacks. The faint or fit occurred on the first day of the illness in two cases, while the latest was on the tenth day. Cardiograms showed nodal rhythm immediately before and after the attack in one; complete heart block during the attack and two hours after the attack respectively in two; sinus tachycardia and multiple auricular extrasystoles in one a few minutes after an attack; and sinus tachycardia between attacks in one. In four cases the site of the infarction was posterior; in the remaining one it could not be determined. Chest pain was moderate and of typical distribution in four cases, but in one it was severe and confined to an area below the left scapula throughout the illness. All five patients died within 29 days of the onset of the infarction. Previous anginal attacks had been experienced by two of them.

TABLE II

Syncope in the Course of Acute Cardiac Infarction

| Case No., Sex, and Age | Attack | Rhythm Recorded | Site of Infarction (*above*) and Degree of Pain (*below*) | Result |
|---|---|---|---|---|
| 11. F./79 | Stokes-Adams (1st day). | Nodal rhythm (before and after). | Posterior. Moderate (chest). | Died 1st day. |
| 12. M./67 | Fainting (1st day). | C.H.B. (2 hours after attack). | Posterior. Moderate (chest). | Died 1st day. |
| 13. M./69 | Stokes-Adams (10th day). | C.H.B. (in attack). | ? Moderate (chest). | Died 29th day. |
| 14. M./69 | Fainting (3rd day). | Sinus tachycardia and Aur. P.C. (after attack). | Posterior. Moderate (chest). | Died 4th day. |
| 15. M./62 | Stokes-Adams (5th day). | Sinus tachycardia (between attacks). | Posterior. Severe (back). | Died 10th day. |

C.H.B.—Complete heart block. Aur. P.C.—Auricular premature contractions.

*Case* 11. A woman of 79 with old-standing hypertension, but a normal cardiogram five years before, and no cardiac symptoms. Retrosternal pain one morning through to the back with numbness of the forearm and wrists, lasted until relieved by morphia five hours later. Heart rate then 90 regular; B.P. 220/110. In after-

noon speech incoherent but had recovered by evening. Fig. 5, taken ten hours after onset, showed nodal rhythm, intraventricular block, and posterior infarction. Immediately after this curve had been taken, the patient became strange in manner; and vomiting, pallor, and stupor followed, and then a sudden brief tonic contraction of all limbs. The patient was then quite unconscious and cold and clammy, and the radial pulse could not be felt. The electrodes were still attached, and Fig. 6 showing

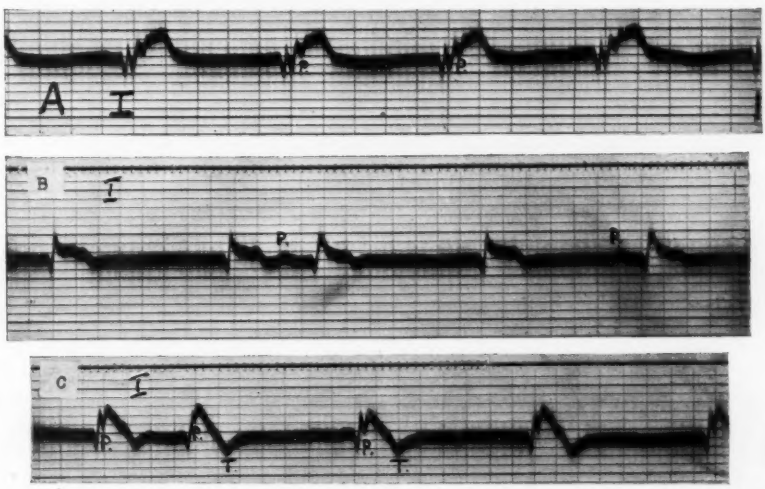



FIG. 6.—Case 11. (A) Nodal rhythm, rate 60; QRS, W-shaped; elevation of RS–T. (B) Taken ten minutes after (A). Change in the form of QRS; RS–T elevation less, with a very small negative T wave; a shallow negative P wave in the RS–T segment; the third and fifth cycles represent sinus beats with prolonged P–R interval.

(C) Taken after breathing had stopped. Nodal rhythm with one premature beat, increased upward deviation of RS–T and deeper inversion of T; rate about 40 a minute.

nodal rhythm was taken immediately. The rate continued to decrease and the voltage of the complexes to diminish until they finally ceased 25 minutes after the onset of the attack.

*Case* 12. A man of 67 woke at 6 a.m. with pain in the mid-chest radiating into both arms, neck, and jaw. He stayed in bed and at 3 p.m. was found unconscious. When seen by his doctor soon after, there was pallor, pulse imperceptible, heart irregular, rate 120. After a short time the pulse could be felt with a rate of 56. At 5 p.m. his skin was pale yellow and moist; some dyspnœa, cervical veins engorged; abdomen a little distended; apex beat heaving in anterior axillary line; heart sounds very distant; B.P. 100/60. Complete heart block (Fig. 7). Death occurred the same day. There was a history in this case of anterior infarction 5 years before and of paroxysmal dyspnœa on and off for 4 years.

*Case* 13. A man of 69 with effort angina for 18 months and a prolonged attack of rest angina 8 months previously. On the tenth day of a second attack of prolonged rest angina, Stokes-Adams attacks began and recurred at intervals of a few minutes. Between the seizures the pulse was irregular, rate about 100; during them the radial pulse could not be felt but as consciousness was regained the beating was resumed at the rapid rate. Fig. 8 taken between attacks shows partial heart block, and the lower strip of lead III taken during a Stokes-Adams attack shows complete heart block. Pulmonary congestion developed and death occurred immediately after a Stokes-Adams attack 19 days later.

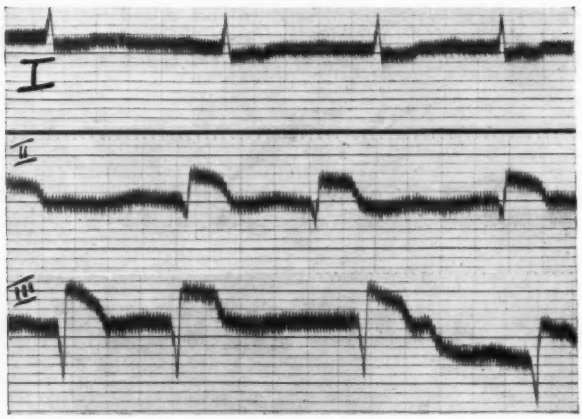



FIG. 7.—Case 12. Complete heart block with an occasional premature beat; ventricular rate 56; changes in QRS, RS–T, and T, suggesting posterior infarction.

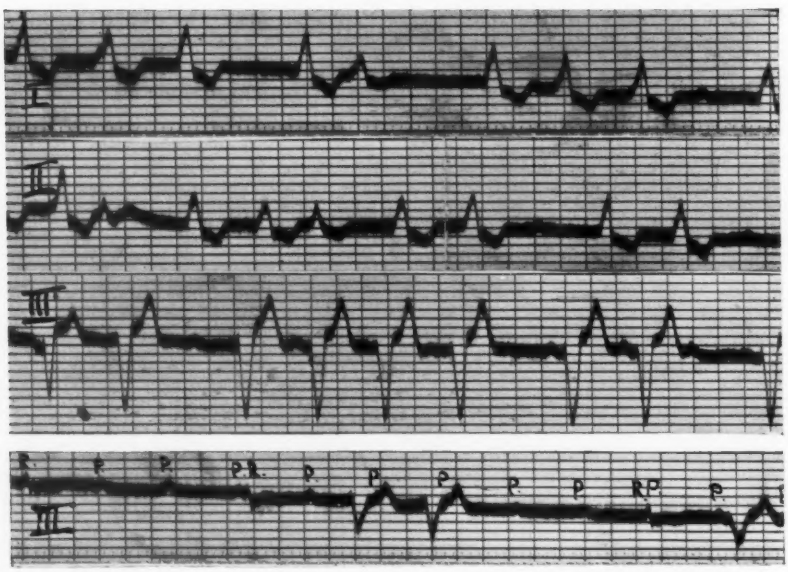



FIG. 8.—Case 13. Partial heart block and intraventricular block; A about 110; V about 100; occasional ventricular extrasystoles. The lower strip of lead III taken during a Stokes-Adams attack shows complete heart block; A, 110; V, 34; QRS and T very low voltage: the third, fourth, and sixth ventricular complexes represent a response to supraventricular impulses or alternatively ventricular extrasystoles.

*Case* 14. A man of 69 after three days of pain in the chest and left arm had repeated brief fainting attacks. There was recurrent vomiting but little dyspnœa; apex beat heaving in the anterior axillary line; radial pulse barely palpable, systolic murmur at the apex. Cardiogram on the third day showed sinus tachycardia with short runs of auricular extrasystoles; rate 135 per minute; RS–T depression in lead I, elevation in lead III; T positive in leads I and IVR, inverted in leads II and III; Q of 4 mm. amplitude in lead III. Posterior infarction. Death on the fourth day.

*Case* 15. A man of 62: slight chest pain for some months and pain below left scapula for five days. Pulse 120, completely irregular. Two days later dyspnœa, vomiting, irregular tachycardia, and persistence of the back pain which became worse while being examined, and the pulse was felt to become progressively slower and more feeble; there was extreme pallor, respiration stopped, and there was a generalized convulsion lasting a few seconds. Consciousness was regained immediately, the pulse returning with a rate of 108 and still completely irregular. The back pain was again distressing and was not controlled until 3/4 grain morphia had been given within half an hour. Slight pyrexia. B.P. 150/95, apex beat not palpable; no signs of failure. Cardiogram 8 hours later showed normal rhythm, rate 110 a minute; P–R interval less than 0 2 sec.; $T_1$ just positive, $T_2$ flat, $T_3$ just negative. Next day another Stokes-Adams attack, again preceded by intense pain in back. Some blood-stained sputum. On succeeding days further Stokes-Adams attacks and death on tenth day. Two other cardiograms taken between the attacks also showed sinus tachycardia with normal A-V conduction; there were progressive changes in the QRS complexes and T waves, consistent with posterior infarction.

## Discussion

The syncope that sometimes marks the onset of cardiac infarction is generally accompanied by severe shock or peripheral circulatory failure, with an abnormally slow heart rate. It may come on very abruptly so that the patient has no warning before he falls to the ground. Anginal pain may be mild or absent throughout. Congestive failure follows, not infrequently, after a short interval. The prognosis in cardiac infarction with syncopal onset, judging from the ten cases reported here, among which there were six deaths within three months, is much graver than usual. However, the average age of these fatal cases was advanced, and the mortality of acute cardiac infarction in the elderly is very high. To produce the sudden syncope of the onset several factors are probably operating, but the principal one must be a nervous reflex leading to vasomotor depression and vagal stimulation causing bradycardia. A subsidiary effect is the reduced force and efficiency of systole that results from the inactivity of the infarcted area of myocardium. There is also the possibility of a direct ischæmic effect on the auricles causing bradycardia and abnormal rhythms. If so, it would fit in with the infarction being more often of the posterior type (excluding one case with ventricular tachycardia there were six posterior and three anterior infarctions), since according to Gross (1921) the right coronary artery takes a larger share in the arterial supply to the auricles. Further support for such an effect is the finding, both at the time of the attacks and later, of depression of the pacemaker and of auricular fibrillation and flutter. Cushing *et al.* (1942) found an abnormal auricular mechanism in 17 of 23 cases of auricular infarction. Finally the influence of age must be considered. This, when it reduces the elasticity of the arterial tree and when it is accompanied by degenerative vascular changes in the brain, must sensitize the cerebrum to any reduction in blood flow. Severe general

and focal cerebral symptoms in cardiac infarction, in the absence of any acute lesion in the arteries of the brain, have been described (Bean & Read, 1942).

Of the five cases here reported with syncope or fit occurring after the onset of cardiac infarction, it was possible to establish the mechanism of the heart beat during the actual attack in one only (Case 13): in this a Stokes-Adams attack coincided with a period of complete heart block, the ventricular rate falling from 100 to 34; cardiac standstill did not occur. As the attack terminated the original rhythm and rate was resumed. In Case 12 complete block was present two hours after a syncopal attack, and although an irregular tachycardia had been noted immediately after the attack, this does not exclude the possibility that it was determined by block, as Case 13 shows. The incidence of complete block in cardiac infarction has been reported as 2 to 3 per cent (Kerr, 1937), but only a proportion of these have Stokes-Adams attacks or even syncopal symptoms.

Nodal rhythm was recorded in Case 11 immediately before a Stokes-Adams seizure, and was again recorded as soon as the convulsion had subsided though consciousness was not recovered and death occurred a few minutes later. A cerebral vascular complication, especially hæmorrhage, has to be considered here, but a further coronary occlusion with acute peripheral circulatory failure would fit in with the change in the ventricular complexes of the cardiogram after the attack.

In Case 14 syncopal attacks appeared on the third day of a cardiac infarct in a man who had slowly passed into a shocked condition. There was sinus tachycardia and short runs of auricular premature beats, the last suggesting that syncope was dependent on auricular paroxysmal tachycardia.

The Stokes-Adams attacks in Case 15 were peculiar in being preceded by great intensification of anginal pain which was located below the left scapula, the pulse at the same time becoming progressively weaker and slower. Cardiograms taken on three separate days between attacks all showed sinus tachycardia with normal A-V conduction, providing no clue to the mechanism involved. This might have been paroxysmal heart block with ventricular standstill except that in this condition the ventricle usually ceases abruptly without preliminary change in heart rate or force. (Parkinson, Papp, and Evans, 1941; Cookson, unpublished observation.) But in any attempt at an explanation the significance of the pain that heralded the attack must be taken into account and it may be that nervous reflexes arising in the heart were largely responsible. On this view the mechanism would be similar to that suggested for the initial syncopal symptoms.

No patient with syncope or Stokes-Adams seizures survived many days, indicating a prognosis even more serious than in the first group.

## Summary

Among 200 patients with acute cardiac infarction, a syncopal or epileptiform attack was observed in 15. At the onset syncope occurred in ten, of whom five were aged 70 or more. They presented the appearance of severe

peripheral circulatory failure, often combined with a slow heart rate but in one there was ventricular tachycardia. Pain might be absent or slight. In five the cardiogram showed abnormal rhythms of supraventricular origin. Posterior infarction was commoner than anterior. Six of the patients had died by the fourth month. Possible causes of the syncope are briefly discussed.

Syncope and fits in the course of cardiac infarction are reported in five patients whose average age was 69. Two had suffered previously from anginal pain. Two had syncope and three had Stokes-Adams attacks. All five died within 29 days of the onset. Abnormal rhythms were recorded in four. Cardiograms during the actual attack showed complete heart block in one, and nodal rhythm with a change in the ventricular complex immediately after a short convulsion in another. Indirect evidence as to the mechanism underlying the attacks in the remaining cases is given.

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# PROCEEDINGS OF THE CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

The SIXTH ANNUAL GENERAL MEETING of the Cardiac Society of Great Britain and Ireland was held in the School of Mineralogy Lecture Theatre, at Cambridge, on Friday, April 10, 1942.

CHAIRMAN: L. B. COLE

44 Members, 4 Temporary Members, and 8 Visitors were present.
The Chairman took the chair at 10.15 a.m.

## PRIVATE BUSINESS

1. The minutes of the last meeting having been printed in the Journal were confirmed and signed.
2. The accounts, audited by Bramwell and Gibson, were presented by the Council and approved, the balance being £61 1*s.* 11*d.*
3. The following two new Members were elected :—

   E. J. Wayne, Sheffield, (Ordinary Member).
   Morgan Jones, Manchester, (Associate Member).

   Ten of the present Associate Members were re-elected for a further period of three years.
4. Terence East and William Evans were elected Members of the Council for the years 1942–46. Gilchrist was appointed to act as a Substitute Member on the Council, so long as Hill (1940–44) was abroad on service.
5. William Evans was appointed to act as Assistant Secretary for the year, and Shirley Smith was thanked for his services during the previous three years.
6. The Society decided that as a war measure the Council should have the power to invite certain members of the Empire Forces or other visitors interested in cardiology to become temporary members for the year, such members being supernumerary to the numbers allowed by the rules and not to pay any subscription.
7. The Secretary reported that :—
   (*a*) since the last meeting he had learnt with regret of the death of Graham Steell, another of the Honorary Members, and that Bramwell had written a notice for the British Heart Journal.
   (*b*) the Committee appointed last year to consider the appointment of members from other parts of the British Empire, had reported that owing to war-time difficulties it was not thought a good time to have any fresh developments in this direction, but that the present situation would best be met by the appointment of temporary members as had now been decided.
   (*c*) the position of the British Heart Journal was satisfactory and the circulation well maintained; and the printers had managed to produce the April 1941 number almost at the right date, in spite of the serious damage done at their works shortly before, and were meeting all the difficulties caused by the war most satisfactorily.

## Discussion on Cardiac Defects Found at Recruiting Medical Boards (*Morning Session*)

Starling gave his experiences as Chairman of a Medical Board. He spoke on the difficulty in the diagnosis of early endocardial lesions, especially by medical officers who were not primarily interested in cardiology. The presence of the functional systolic murmur which disappeared on full inspiration and often with a change of posture still occasioned much doubt in the minds of some members of the board. During two and a half years he had supervised the rejection of about 50 cardiac cases from the Services and 24 of these were cases of early mitral stenosis or aortic regurgitation. None of them had any symptoms nor did a number of them give a story of a past rheumatic infection. Starling also called attention to the confusion that existed at Medical Boards and also in the profession regarding the estimation of blood pressure and the interpretation of a raised one, and he particularly emphasized the need to take three readings before labelling a recruit as showing a raised blood pressure.

## Short Communications

### Cardiac Diagnosis in Recruits

John Parkinson presented some observations arising from routine examination of 500 candidates referred by recruiting medical boards for a cardiological examination because of suspected cardiac defects. Experience of diagnosis in civil life was gained mostly on developed disease with symptoms, whereas wartime provided a distinct opportunity for studying and codifying the first signs of early and symptomless cardiac disease. There was a wealth of borderline cases. Textbooks gave the characteristic rather than the earliest diagnostic signs, and comprehensive diagnosis might better be provided as (*a*) early diagnosis, (*b*) established diagnosis, and (*c*) differential diagnosis.

*Aortic Incompetence* was twice as common as mitral stenosis in these recruits. The apex-beat was affected in force or position in only half of the 50 clear cases, and then it rarely reached the sixth interspace. In half there was a mitral systolic murmur, which too often satisfied an examiner as being primarily mitral, but should certainly raise the possibility of a causative aortic incompetence. A loud aortic second sound might obscure the diastolic murmur. The blood pressure was valueless in the slighter cases. Left axis deviation was commonly absent, though $T_2$ and $T_3$ inversion was recorded with it in three cases, and $T_1$ inversion in none. Cardioscopy (X-ray screening of the heart) showed increased convexity of the left ventricle in most, and this was nicely confirmed in the left oblique position. Hearing the aortic diastolic murmur and seeing the enlarged left ventricle were the foundations of an early diagnosis, and attention should be concentrated on these.

*Mitral Stenosis* was studied in 25 early cases. There was no thrill, though the apex beat might be sudden in quality. A short presystolic murmur was heard in 15, but in 9 of these only immediately on lying on the left side after exertion. This invaluable technique was a bequest from the last world war. A sharp loud first sound at the apex is in itself insufficient, but a long loud and constant mitral systolic murmur needs only radiological support to establish the diagnosis. In the anterior view there may be a slight extension outwards of the left border of the heart in the conus region, not so much as a convexity—merely a broadening of the heart at the conus level. With this or without, in the right oblique view the left auricle (atrium) is seen to be enlarged because the opacified œsophagus *changes direction* backwards at a high level—at the top of the auricle. This early sign may precede any obvious increase in the

convexity of the auricle as a whole. Right axis deviation or P wave changes in the electrocardiogram are seldom of assistance in early diagnosis, both being absent in the majority.

*Early High Blood Pressure.* There were 18 cases with a blood pressure above 160/90, and an average of 175/100. Eight were aged 20–30 years, ten were aged 31–40. The apex beat was displaced or forcible (or both) in half the cases, and a mitral systolic murmur was noticeable also in half. Cardioscopic support was obtained in more than half the cases, the left ventricle being enlarged and unduly convex. Electrocardiographic evidence was almost valueless.

*Various Cardiac Defects.* There were 20, i.e. 4 per cent, with congenital lesions. In 30 healthy cases, it was simply the well-known apparent displacement of the apex-beat from slight scoliosis that led the medical board to suspect enlargement. Nervous tachycardia was the feature in 30 cases. There were 8 cases with slight thyroid enlargement, mostly without tachycardia. Coronary thrombosis had occurred in three recruits aged 31, 32, and 35 respectively.

*In conclusion*, attention was directed to :—

(1) The need for routine radioscopy by the cardiologist, and its usefulness in early diagnosis.

(2) The restricted value of the electrocardiograph for this purpose; only in doubtful arrhythmia or precocious anginal pain was it applicable to recruiting medical problems.

(3) A duty of the Cardiac Society to take a leading part after the war in pressing for national research on rheumatic fever in childhood.

## The Electrocardiogram in Friedreich Disease

William Evans

(Published in full; p. 91)

## Fainting and Fits in Cárdiac Infarction

Harold Cookson

Apart from pain of great intensity, cerebral vascular complications, and sudden death, which might be regarded as the extreme form of syncopal attack, disturbances of consciousness had been noted in a small proportion of cases of acute cardiac infarction. The chief clinical features of 13 such cases were given. Faintness or loss of consciousness had occurred as the initial symptom in 9 and early examination had shown the picture of peripheral circulatory failure combined with bradycardia in most. Of 4 cases with disturbance of consciousness after the onset, three had Stokes-Adams attacks, on the first, fifth, and tenth days respectively ; in the fourth there was fainting on the third day associated with sinus tachycardia and short runs of auricular premature beats.

Cardiograms taken at the time of the attacks or at varying intervals thereafter had shown a disturbance of rhythm in the majority. All the common varieties of abnormal rhythm had been recorded. The site of infarction was nearly always posterior. Pain might be completely absent. Eight of the 13 patients had died within six weeks.

To account for the picture of peripheral circulatory failure combined with bradycardia seen in those with syncopal symptoms at the onset it was suggested that the factors were: (1) reflex vaso-motor depression and vagal stimulation; (2) a direct mechanical effect of the infarct on cardiac output; and (3) possibly ischæmia of the auricles; The last would fit in with the observation that the infarct was usually of the posterior type and also with the occurrence of rhythms depending on depression of the pacemaker and of auricular flutter and fibrillation.

(Published in full; p. 163)

### Inversion of T Waves after Attacks of Angina Pectoris

Donald Hall and Kemball Price (*introduced*)

Kemball Price described a case in which inversion of the T wave in leads I and IV followed an attack of angina pectoris but only persisted for twelve days. The T wave in lead IV again became inverted after a further anginal attack and was directed upwards following amyl nitrate inhalation. Two months later the cardiographic changes remained permanent after prolonged pain; at necropsy cardiac infarction was found present. He suggested that during the first two attacks there might have been an interference with the blood supply to a portion of a muscle bundle, but short of infarction from occlusion of a main coronary artery.

After luncheon a demonstration was given on Hypertension of Renal Origin by E. B. Verney, F. J. Lockett, and W. J. O'Connor (*all introduced*). Several dogs were shown in different stages where operations had been performed so that the blood-pressure could be measured in arterial loops and the blood supply to the kidney clamped in an accurate manner.

### Discussion on the Importance of the Cardiovascular System in Aviation Medicine

Rook said that in *The Medical Examination of Candidates for Flying Training* too much stress can be placed on tests of cardiovascular efficiency. So long as there is no organic lesion of heart or vessels and the pulse rate and blood pressure readings are within fairly wide limits, most candidates can be accepted, whatever their reactions to the various tests, for it has been shown that proper training will soon bring their physical efficiency to a high level. Of all the factors necessary to make a war-time pilot, temperament is the most important; physical efficiency can be improved, temperament is largely unalterable, at any rate within a short period.

*Operational Flying* throws a strain on the cardiovascular system chiefly in two ways, firstly owing to the effect of altitude and secondly as the result of variations in speed. *Anoxia* begins to show its effect fairly early in an ascent causing, at first, increase in the pulse rate and in the pulse pressure. The severity of the effects depends on certain variable factors : (1) the altitude to which the ascent is made; (2) the duration of stay at altitude; and (3) the rate of ascent. In pilots acclimatization to altitude such as occurs in mountaineers is almost unknown and permanent changes in the size of the heart or in the blood are rarely seen. Actually deterioration is more likely to occur than acclimatization. The normal cardiovascular system can withstand anoxia better than most of the other systems of the body, and the circulation may still be going on when respiration and all reflexes have ceased. With the damaged heart anoxia is a potent factor in producing symptoms and may lead to failure.

The effects of *Acceleration and Deceleration* are especially of importance to the war-time pilot. These effects are met with chiefly while caterpulting or during turns at high speeds. The resultant forces may cause symptoms by their action on the more fluid parts of the body, principally the blood. On the arterial side these forces interfere with the flow of the blood, while on the venous side they prevent return to the auricles and lead to inadequate filling of the ventricles. In most instances the forces act from the head towards the legs (positive accelerations) producing an anæmia of the brain and causing symptoms that include temporary blindness and possibly actual loss of consciousness. If the forces act from the legs towards the head (negative accelerations) intense engorgement of the cerebral vessels and severe symptoms may result. Transverse accelerations, which act across the long axis of the body, affect the cardiovascular system least. It is possible by bracing the abdominal muscles and bending

forward so that the forces act partly transversely to counteract the effect of positive accelerations to some extent.

SQUADRON LEADER W. K. STEWART, A.F.C. (*introduced*), from the Royal Air Force Physiological Laboratory, demonstrated a film showing the effect of positive acceleration occurring during actual flight.

### HEMIPLEGIA IN CYANOTIC CONGENITAL HEART DISEASE

BRUCE PERRY

(Published in full; p. 121)

### SYPHILITIC ANGINA PECTORIS

EVAN BEDFORD AND EVAN JONES

(To be published in full)

### LIGATURE OF SIX CASES OF INFECTED DUCTUS ARTERIOSUS

BOURNE reported six cases (the first already reported in this Journal, Vol. 3, 228, 1941), all of whom survived the operation by six weeks or more, and five of whom completely recovered; one case with a mycotic aneurysm died showing a continuation of the infection. One was alive 27 months and another 17 months after the operation. Four were back at full work. Chemotherapy was used as an adjunct to ligature, but that ligation was the effective factor was proved by the fact that it produced cure when chemotherapy failed or when chemotherapy was withheld. In all cases the diastolic pressure increased greatly after the operation but became lowered to the normal after a period of six weeks or so. The systolic pressure was increased to a lesser extent. The heart in the only case in which accurate pre-operative measurement on cardioscopy was possible was found to be 2 cm. less in its transverse diameter 17 months after the operation; the previously enlarged pulmonary conus had disappeared. In this case and in one other the typical systolic-diastolic murmur returned after an interval and the cause of this was obscure because the blood pressure and heart outline continued to be normal.

### CHEST LEAD, $CR_7$, IN POSTERIOR CARDIAC INFARCTION

A. HUNTER (*introduced*) AND WILLIAM EVANS mentioned the difficulties in diagnosis between inversion of the T wave in lead III in posterior cardiac infarction and in other conditions, either in health or disease. He showed the results obtained from the use of a chest lead ($CR_7$) in which an exploring electrode was placed in the left posterior axillary line and paired with one on the right arm. Changes towards inversion of the T wave in this lead appeared in the majority of cases of cardiac infarction although it was shown as well in the limb leads, but its importance was found in confirming the physiological nature of inversion of the $T_3$ in health for the $CR_7$ was always upright in this circumstance. Apart from assisting in the diagnosis of physiological inversion of the $T_3$ from the inverted $T_3$ of cardiac infarction, the chest lead $CR_7$ was also found to aid the recognition of the cardiogram of pericardial disease which might suggest posterior cardiac infarction.

(To be published in full.)

### The Return of Normal Rhythm After Total Thyroidectomy for Mitral Stenosis and Auricular Fibrillation

Cookson said that occasional cases had been reported, suggesting that the secretion of the normal thyroid gland might lead to the development of auricular fibrillation in non-goitrous heart disease. In two cases seen by Cookson, in which toxic goitre, mitral stenosis, and auricular fibrillation had been combined, total thyroidectomy had been carried out, and in each case the rhythm had returned to normal spontaneously. An account was given of the clinical condition and autopsy findings in a woman with mitral stenosis, auricular fibrillation, and recurrent failure. Her whole thyroid had been removed, and three or four months after the operation, when signs of myxœdema were obvious, normal rhythm was found to have returned. There were then no signs of failure and her effort tolerance had improved. The radiogram, however, showed considerable increase in the cardiac silhouette and resembled in shape that of pericardial effusion. The patient remained well for four months. Symptoms and signs of failure then appeared but the pulse remained regular. She died after a week's illness; necropsy showed advanced mitral stenosis and the pericardium contained 24 oz. of clear yellow fluid. It was considered probable that severe myxœdema had played a part in the final breakdown, particularly through the effect of a large pericardial effusion.

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The dinner was held at Caius by kind permission of the Bursar. Emmanuel proposed the health of the Society.

Bramwell proposed the health of the guests, including the Master and the President of the College of Physicians. The Master replied on behalf of the guests.

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